10554187

Connecting via Winsock to STN

Welcome to STN International! Enter x:x

LOGINID:ssspta1612bxr

PASSWORD:
TERMINAL (ENTER 1, 2, 3, OR ?):2

* * * * * * * * * * Welcome to STN International * * * * * * * * * *

NEWS	1		Web Page URLs for STN Seminar Schedule - N. America
NEWS	2		"Ask CAS" for self-help around the clock
NEWS	3	FEB 27	New STN AnaVist pricing effective March 1, 2006
NEWS	4	MAY 10	CA/CAplus enhanced with 1900-1906 U.S. patent records
NEWS	5	MAY 11	KOREAPAT updates resume
NEWS	6	MAY 19	Derwent World Patents Index to be reloaded and enhanced
NEWS	7	MAY 30	IPC 8 Rolled-up Core codes added to CA/CAplus and USPATFULL/USPAT2
NEWS	8	MAY 30	The F-Term thesaurus is now available in CA/CAplus
NEWS	9	JUN 02	The first reclassification of IPC codes now complete in INPADOC
NEWS	10	JUN 26	TULSA/TULSA2 reloaded and enhanced with new search and and display fields
NEWS	11	JUN 28	Price changes in full-text patent databases EPFULL and PCTFULL
NEWS	12	JUl 11	CHEMSAFE reloaded and enhanced
NEWS	13	JUl 14	FSTA enhanced with Japanese patents
NEWS	14	JUl 19	Coverage of Research Disclosure reinstated in DWPI
NEWS	15	AUG 09	INSPEC enhanced with 1898-1968 archive
NEWS	16	AUG 28	ADISCTI Reloaded and Enhanced
NEWS	17	AUG 30	CA(SM)/CAplus(SM) Austrian patent law changes
NEWS	18	SEP 11	CA/CAplus enhanced with more pre-1907 records
NEWS	19	SEP 21	CA/CAplus fields enhanced with simultaneous left and right truncation
NEWS	20	SEP 25	CA(SM)/CAplus(SM) display of CA Lexicon enhanced
NEWS	21	SEP 25	CAS REGISTRY(SM) no longer includes Concord 3D coordinates
NEWS	22	SEP 25	CAS REGISTRY(SM) updated with amino acid codes for pyrrolysine
NEWS	23	SEP 28	CEABA-VTB classification code fields reloaded with new classification scheme
NEWS	24	OCT 02	MARPAT(R) now updated daily

NEWS EXPRESS JUNE 30 CURRENT WINDOWS VERSION IS V8.01b, CURRENT MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP), AND CURRENT DISCOVER FILE IS DATED 26 JUNE 2006.

NEWS HOURS	STN Operating Hours Plus Help Desk Availability
NEWS LOGIN	Welcome Banner and News Items
NEWS IPC8	For general information regarding STN implementation of IPC 8
NEWS X25	X.25 communication option no longer available

Enter NEWS followed by the item number or name to see news on that specific topic.

All use of STN is subject to the provisions of the STN Customer

Updated Search

agreement. Please note that this agreement limits use to scientific research. Use for software development or design or implementation of commercial gateways or other similar uses is prohibited and may result in loss of user privileges and other penalties.

* * * * * * * * * * * * * * * * STN Columbus * * * * * * * * * * * * * * * * *

FILE 'HOME' ENTERED AT 17:51:52 ON 02 OCT 2006

=> file reg

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	0.21	0.21

FILE 'REGISTRY' ENTERED AT 17:52:01 ON 02 OCT 2006
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.
PLEASE SEE "HELP USAGETERMS" FOR DETAILS.
COPYRIGHT (C) 2006 American Chemical Society (ACS)

Property values tagged with IC are from the ZIC/VINITI data file provided by InfoChem.

STRUCTURE FILE UPDATES: 1 OCT 2006 HIGHEST RN 909214-11-5
DICTIONARY FILE UPDATES: 1 OCT 2006 HIGHEST RN 909214-11-5

New CAS Information Use Policies, enter HELP USAGETERMS for details.

TSCA INFORMATION NOW CURRENT THROUGH June 30, 2006

Please note that search-term pricing does apply when conducting SmartSELECT searches.

REGISTRY includes numerically searchable data for experimental and predicted properties as well as tags indicating availability of experimental property data in the original document. For information on property searching in REGISTRY, refer to:

http://www.cas.org/ONLINE/UG/regprops.html

=> s akt or ? akt() oncogene? () protein? or akt () kinase () transform? () protein?
ADDITIONAL CHARACTERS REQUIRED AFTER '?' FOR LEFT TRUNCATION
Additional characters must follow the left truncation symbol in your search term. If your search term contains a punctuation mark before the truncation symbol and you are searching in a field that uses implied proximity, the system may interpret the truncation symbol as being at the beginning of a term. Implied proximity is used in search fields indexed as single words, for example, the Basic Index. To see which fields in the current file have left truncation, enter "HELP SFIELDS" at an arrow prompt (=>).

=> file hcaplus

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	1.32	1.53

FILE 'HCAPLUS' ENTERED AT 17:54:03 ON 02 OCT 2006
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.
PLEASE SEE "HELP USAGETERMS" FOR DETAILS.
COPYRIGHT (C) 2006 AMERICAN CHEMICAL SOCIETY (ACS)

Updated Search

10554187

FILE COVERS 1907 - 2 Oct 2006 VOL 145 ISS 15
FILE LAST UPDATED: 1 Oct 2006 (20061001/ED)

New CAS Information Use Policies, enter HELP USAGETERMS for details.

This file contains CAS Registry Numbers for easy and accurate
substance identification.

```
=> s akt or ?akt () oncogene? () protein? or akt () kinase () transform? () protein?
         11149 AKT
            18 AKTS
         11157 AKT
                 (AKT OR AKTS)
         12944 ?AKT
         35156 ONCOGENE?
       2277690 PROTEIN?
             0 ?AKT (W) ONCOGENE? (W) PROTEIN?
         11149 AKT
            18 AKTS
         11157 AKT
                 (AKT OR AKTS)
        273798 KINASE
         53395 KINASES
        282501 KINASE
                 (KINASE OR KINASES)
        640626 TRANSFORM?
       2277690 PROTEIN?
             0 AKT (W) KINASE (W) TRANSFORM? (W) PROTEIN?
L1       11157 AKT OR ?AKT (W) ONCOGENE? (W) PROTEIN? OR AKT (W) KINASE (W)
               TRANSFORM? (W) PROTEIN?

=> s l1 () inhibit?
       1866618 INHIBIT?
L2         383 L1 (W) INHIBIT?

=> s l2 and review/dt
       1962466 REVIEW/DT
L3          17 L2 AND REVIEW/DT

=> s l3 and cancer
        292545 CANCER
         42708 CANCERS
        303633 CANCER
                 (CANCER OR CANCERS)
L4           8 L3 AND CANCER

=> d l4, ibib abs hitstr, 1-8
```

Updated Search

10554187

L4 ANSWER 1 OF 8 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2006:350627 HCAPLUS
DOCUMENT NUMBER: 144:465166
TITLE: Akt Signaling and Cancer: Surviving but not Moving On
AUTHOR(S): Toker, Alex; Yoeli-Lerner, Merav
CORPORATE SOURCE: Department of Pathology, Beth Israel Deaconess Medical Center, Harvard Medical School, Boston, MA, USA
SOURCE: Cancer Research (2006), 66(8), 3963-3966
CODEN: CNREA8; ISSN: 0008-5472
PUBLISHER: American Association for Cancer Research
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
AB A review. The frequent deregulation of the phosphoinositide 3-kinase/Akt survival signaling pathway in cancer has prompted significant interest in blocking this pathway to treat cancer. Recently, however, two studies have shown that the Akt isoform Akt1 limits the invasive migration of breast cancer cells. These studies suggest that Akt1 may have a dual role in tumorigenesis, acting not only pro-oncogenically by suppressing apoptosis but also anti-oncogenically by suppressing invasion and metastasis. We discuss the possible implications of these findings for therapeutic development of Akt inhibitors to treat cancer.
REFERENCE COUNT: 28 THERE ARE 28 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 2 OF 8 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2005:1349832 HCAPLUS
DOCUMENT NUMBER: 144:307393
TITLE: Molecular strategies targeting the host component of cancer to enhance tumor response to radiation therapy
AUTHOR(S): Kim, Dong Wook; Huamani, Jessica; Fu, Allie; Hallahan, Dennis E.
CORPORATE SOURCE: Department of Radiation Oncology, Vanderbilt Ingram Cancer Center, Nashville, TN, USA
SOURCE: International Journal of Radiation Oncology, Biology, Physics (2005), Volume Date 2006, 64(1), 38-46
CODEN: IOBPD3; ISSN: 0360-3016
PUBLISHER: Elsevier Inc.
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
AB A review. The tumor microenvironment, in particular, the tumor vasculature, as an important target for the cytotoxic effects of radiation therapy is an established paradigm for cancer therapy. The authors review the evidence that the phosphoinositide 3-kinase (PI3K)/Akt pathway is activated in endothelial cells exposed to ionizing radiation (IR) and is a mol. target for the development of novel radiation sensitizing agents. On the basis of this premise, several promising preclin. studies that targeted the inhibition of the PI3K/Akt activation as a potential method of sensitizing the tumor vasculature to the cytotoxic effects of IR have been conducted. An innovative strategy to guide cytotoxic therapy in tumors treated with radiation and PI3K/Akt inhibitors is presented. The evidence supports a need for further investigation of combined-modality therapy that involves radiation therapy and inhibitors of PI3K/Akt pathway as a promising strategy for improving the treatment of patients with cancer.
REFERENCE COUNT: 68 THERE ARE 68 CITED REFERENCES AVAILABLE FOR THIS

Updated Search

10554187

RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 3 OF 8 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2005:1211155 HCAPLUS
DOCUMENT NUMBER: 144:16324
TITLE: The Akt/PKB pathway: molecular target for cancer drug discovery
AUTHOR(S): Cheng, Jin Q.; Lindsley, Craig W.; Cheng, George Z.; Yang, Hua; Nicosia, Santo V.
CORPORATE SOURCE: Departments of Pathology and Interdisciplinary Oncology, H Lee Moffitt Cancer Center and Research Institute, University of South Florida College of Medicine, Tampa, FL, 33612, USA
SOURCE: Oncogene (2005), 24(50), 7482-7492
CODEN: ONCNES; ISSN: 0950-9232
PUBLISHER: Nature Publishing Group
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
AB A review. The serine/threonine kinase Akt/PKB pathway presents an exciting new target for mol. therapeutics, as it functions as a cardinal nodal point for transducing extracellular (growth factor and insulin) and intracellular (receptor tyrosine kinases, Ras and Src) oncogenic signals. In addition, alterations of the Akt pathway have been detected in a number of human malignancies. Ectopic expression of Akt, especially constitutively activated Akt, is sufficient to induce oncogenic transformation of cells and tumor formation in transgenic mice as well as chemoresistance. Akt has a wide range of downstream targets that regulate tumor-associated cell processes such as cell growth, cell cycle progression, survival, migration, epithelial-mesenchymal transition and angiogenesis. Blockage of Akt signaling results in apoptosis and growth inhibition of tumor cells with elevated Akt. The observed dependence of certain tumors on Akt signaling for survival and growth has wide implications for cancer therapy, offering the potential for preferential tumor cell killing. In the last several years, through combinatorial chemical, high-throughput and virtual screening, and traditional medicinal chemical, a number of inhibitors of the Akt pathway have been identified. This review focuses on ongoing translational efforts to therapeutically target the Akt pathway.
REFERENCE COUNT: 131 THERE ARE 131 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 4 OF 8 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2005:493934 HCAPLUS
DOCUMENT NUMBER: 143:242080
TITLE: Effects on cell viability
AUTHOR(S): Guzman, M.
CORPORATE SOURCE: Department of Biochemistry and Molecular Biology I, School of Biology, Complutense University, Madrid, 28040, Spain
SOURCE: Handbook of Experimental Pharmacology (2005), 168(Cannabinoids), 627-642
CODEN: HEPHD2; ISSN: 0171-2004
PUBLISHER: Springer GmbH
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
AB A review. Cannabinoids are known to control the cell survival/death decision, leading to different outcomes that depend on the nature of the target cell and its proliferative or differentiation status. Cannabinoids

Updated Search

induce growth arrest or apoptosis in a number of transformed cells in culture. They do so by modulating key cell signaling pathways involved in the control of tumor cell fate. The best-characterized example is cannabinoid-induced apoptosis of glioma cells, which occurs via sustained ceramide accumulation, extracellular signal-regulated kinase activation, and Akt inhibition. In addition, cannabinoid administration inhibits the angiogenesis and slows the growth of different types of tumors in laboratory animals. By contrast, most of the exptl. evidence indicates that cannabinoids protect normal neurons and glial cells from apoptosis as induced by toxic insults such as glutamatergic overstimulation, ischemia, and oxidative damage. It is therefore very likely that cannabinoids regulate cell survival and cell death pathways differently in tumor and non-tumor cells. Regarding immune cells, cannabinoids affect proliferation and survival in a complex and still obscure manner that depends on the exptl. setting. The findings reviewed here might set the basis for the use of cannabinoids in the treatment of cancer and neurodegenerative diseases.

REFERENCE COUNT: 87 THERE ARE 87 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 5 OF 8 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2004:703858 HCAPLUS
DOCUMENT NUMBER: 141:184489
TITLE: AKT: A potential target for thyroid cancer therapy
AUTHOR(S): Kada, Faiza; Saji, Motoyasu; Ringel, Matthew D.
CORPORATE SOURCE: The Washington Hospital Center/MedStar Research Institute, Washington, DC, USA
SOURCE: Current Drug Targets: Immune, Endocrine and Metabolic Disorders (2004), 4(3), 181-185
CODEN: CDTIBT; ISSN: 1568-0088
PUBLISHER: Bentham Science Publishers Ltd.
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English

AB A review. Thyroid cancer is a heterogeneous disorder characterized by gene mutations that activate signaling pathways, and also by abnormalities in tumor suppressor genes and cell cycle proteins. Activation of the Akt/PKB signaling pathway appears to be an important event in thyroid tumorigenesis and, perhaps, in tumor progression too. Akt is activated in Cowden's syndrome through inactivation of PTEN, a neg. regulator of Akt. Cowden's syndrome is an autosomal dominant multiorgan hamartoma syndrome characterized by benign and malignant thyroid tumors, breast cancers, and colon cancers. In addition, the Akt pathway appears to be activated in a significant proportion of sporadic thyroid cancers through activation of growth factor pathways by thyroid oncogenes and/or receptor overexpression. Disruption of PI3-kinase activity pharmacol. or disruption of Akt signaling using dominant neg. cDNA expression have demonstrated salutary effects on several cancer models in vitro. Therefore, Akt represents an attractive target for pharmaceutical development for a variety of malignancies, including thyroid cancer.

REFERENCE COUNT: 72 THERE ARE 72 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 6 OF 8 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2004:604067 HCAPLUS
DOCUMENT NUMBER: 141:199325
TITLE: Hypothesis: cannabinoid therapy for the treatment of

10554187

gliomas?
AUTHOR(S): Velasco, Guillermo; Galve-Roperh, Ismael; Sanchez, Cristina; Blazquez, Cristina; Guzman, Manuel
CORPORATE SOURCE: School of Biology, Department of Biochemistry and Molecular Biology I, Complutense University, Madrid, 28040, Spain
SOURCE: Neuropharmacology (2004), 47(3), 315-323
CODEN: NEPHBW; ISSN: 0028-3908
PUBLISHER: Elsevier B.V.
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
AB A review. Gliomas, in particular glioblastoma multiforme or grade IV astrocytoma, are the most frequent class of malignant primary brain tumors and one of the most aggressive forms of cancer. Current therapeutic strategies for the treatment of glioblastoma multiforme are usually ineffective or just palliative. During the last few years, several studies have shown that cannabinoids-the active components of the plant Cannabis sativa and their derivs.-slow the growth of different types of tumors, including gliomas, in laboratory animals. Cannabinoids induce apoptosis of glioma cells in culture via sustained ceramide accumulation, extracellular signal-regulated kinase activation and Akt inhibition. In addition, cannabinoid treatment inhibits angiogenesis of gliomas in vivo. Remarkably, cannabinoids kill glioma cells selectively and can protect non-transformed glial cells from death. These and other findings reviewed here might set the basis for a potential use of cannabinoids in the management of gliomas.
REFERENCE COUNT: 68 THERE ARE 68 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 7 OF 8 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2004:506589 HCAPLUS
DOCUMENT NUMBER: 141:98885
TITLE: The development of phosphatidylinositol ether lipid analogues as inhibitors of the serine/threonine kinase, Akt
AUTHOR(S): Gills, Joell J.; Dennis, Phillip A.
CORPORATE SOURCE: NCI, Bethesda, MD, 20889, USA
SOURCE: Expert Opinion on Investigational Drugs (2004), 13(7), 787-797
CODEN: EOIDER; ISSN: 1354-3784
PUBLISHER: Ashley Publications Ltd.
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
AB A review. The serine/threonine kinase Akt is a component of the phosphatidylinositol 3'-kinase/Akt signal transduction pathway that is activated by receptor tyrosine kinases, activated Ras and integrins. As Akt regulates many processes crucial to carcinogenesis, and Akt activation has been observed in human cancers, intense efforts are underway to develop Akt inhibitors as cancer therapeutics. Towards this aim, phosphatidylinositol ether lipid analogs (PIAs), which are structurally similar to the products of phosphatidylinositol 3'-kinase, have been synthesized. PIAs inhibit Akt translocation, phosphorylation and kinase activity. Furthermore, they selectively induce apoptosis in cancer cell lines that depend on Akt for survival. This review will trace the development of PIAs, cover the biol. activities of PIAs and discuss future steps and challenges in their development.
REFERENCE COUNT: 59 THERE ARE 59 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

Updated Search

10554187

L4 ANSWER 8 OF 8 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2002:607171 HCAPLUS
DOCUMENT NUMBER: 138:162768
TITLE: Targeting serine/threonine protein kinase B/Akt and cell-cycle checkpoint kinases for treating cancer
AUTHOR(S): Li, Qun; Zhu, Gui-Dong
CORPORATE SOURCE: Cancer Research, Global Pharmaceutical Discovery, Abbott Laboratories, Abbott Park, IL, 60064-6101, USA
SOURCE: Current Topics in Medicinal Chemistry (Hilversum, Netherlands) (2002), 2(9), 939-971
CODEN: CTMCCL; ISSN: 1568-0266
PUBLISHER: Bentham Science Publishers Ltd.
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
AB A review. Over the past decade, protein kinases have emerged as a group of mol. targets with the potential to be "cancer-specific", allowing the selective targeting of cancer cells vs. normal cells. These selective anticancer drugs would eliminate the cytotoxic side effects that are associated with conventional cancer chemotherapy. This article will focus on two emerging and less-explored protein serine/threonine kinase targets: PKB/Akt and checkpoint kinase 1 (Chk1). Protein kinase B/Akts are a group of serine/threonine kinases that are overexpressed in a variety of human tumors. An Akt inhibitor would target the imbalance of pro-vs. anti-apoptosis regulation in cancerous as compared to healthy cells. Thus, a greater therapeutic window than conventional cytotoxic chemotherapy is expected. Cell-cycle checkpoints have become attractive targets since some of them, such as the G1/S checkpoint, are defective in most tumor cells. Inhibition of one or more of the remaining checkpoint(s) could make cancerous cells more sensitive than healthy cells toward DNA damaging agents or radiation therapy. Among the checkpoint kinases, Chk1 appears to be an attractive mol. target. Chk1 blocks the activation of the Cdc2-cyclin B kinase complex, and hence entry into mitosis, by disrupting the translocation of the phosphatase Cdc25C from the cytoplasm to the nucleus. A limited number of small mol. inhibitors in this emerging field and their mode of action will be reviewed.
REFERENCE COUNT: 275 THERE ARE 275 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> d his

(FILE 'HOME' ENTERED AT 17:51:52 ON 02 OCT 2006)

FILE 'REGISTRY' ENTERED AT 17:52:01 ON 02 OCT 2006

FILE 'HCAPLUS' ENTERED AT 17:54:03 ON 02 OCT 2006
L1 11157 S AKT OR ?AKT () ONCOGENE? () PROTEIN? OR AKT () KINASE () TRAN
L2 383 S L1 () INHIBIT?
L3 17 S L2 AND REVIEW/DT
L4 8 S L3 AND CANCER

=> s l3 not l4
L5 9 L3 NOT L4

=> s l5 and hyperinsulinisum?

Updated Search

10554187

0 HYPERINSULINISUM?
L6 0 L5 AND HYPERINSULINISUM?

=> s l4 and insulin?
194576 INSULIN?
L7 1 L4 AND INSULIN?

=> d l7, ibib abs hitstr, 1

L7 ANSWER 1 OF 1 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2005:1211155 HCAPLUS
DOCUMENT NUMBER: 144:16324
TITLE: The Akt/PKB pathway: molecular target for cancer drug discovery
AUTHOR(S): Cheng, Jin Q.; Lindsley, Craig W.; Cheng, George Z.; Yang, Hua; Nicosia, Santo V.
CORPORATE SOURCE: Departments of Pathology and Interdisciplinary Oncology, H Lee Moffitt Cancer Center and Research Institute, University of South Florida College of Medicine, Tampa, FL, 33612, USA
SOURCE: Oncogene (2005), 24(50), 7482-7492
CODEN: ONCNES; ISSN: 0950-9232
PUBLISHER: Nature Publishing Group
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
AB A review. The serine/threonine kinase Akt/PKB pathway presents an exciting new target for mol. therapeutics, as it functions as a cardinal nodal point for transducing extracellular (growth factor and insulin) and intracellular (receptor tyrosine kinases, Ras and Src) oncogenic signals. In addition, alterations of the Akt pathway have been detected in a number of human malignancies. Ectopic expression of Akt, especially constitutively activated Akt, is sufficient to induce oncogenic transformation of cells and tumor formation in transgenic mice as well as chemoresistance. Akt has a wide range of downstream targets that regulate tumor-associated cell processes such as cell growth, cell cycle progression, survival, migration, epithelial-mesenchymal transition and angiogenesis. Blockage of Akt signaling results in apoptosis and growth inhibition of tumor cells with elevated Akt. The observed dependence of certain tumors on Akt signaling for survival and growth has wide implications for cancer therapy, offering the potential for preferential tumor cell killing. In the last several years, through combinatorial chemical, high-throughput and virtual screening, and traditional medicinal chemical, a number of inhibitors of the Akt pathway have been identified. This review focuses on ongoing translational efforts to therapeutically target the Akt pathway.
REFERENCE COUNT: 131 THERE ARE 131 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> d his

(FILE 'HOME' ENTERED AT 17:51:52 ON 02 OCT 2006)

FILE 'REGISTRY' ENTERED AT 17:52:01 ON 02 OCT 2006

FILE 'HCAPLUS' ENTERED AT 17:54:03 ON 02 OCT 2006
L1 11157 S AKT OR ?AKT () ONCOGENE? () PROTEIN? OR AKT () KINASE () TRAN
L2 383 S L1 () INHIBIT?

Updated Search

10554187

```
L3            17 S L2 AND REVIEW/DT
L4             8 S L3 AND CANCER
L5             9 S L3 NOT L4
L6             0 S L5 AND HYPERINSULINISUM?
L7             1 S L4 AND INSULIN?

=> s l4 not l7
L8             7 L4 NOT L7

=> s l5 not l7
L9             9 L5 NOT L7

=> s l9 and hyperproliferative () disorder?
          1693 HYPERPROLIFERATIVE
        434770 DISORDER?
           442 HYPERPROLIFERATIVE (W) DISORDER?
L10          0 L9 AND HYPERPROLIFERATIVE (W) DISORDER?

=> s l9 and angiogenesis?
         34518 ANGIOGENESIS?
L11          1 L9 AND ANGIOGENESIS?

=> d l11, ibib abs, 1

L11  ANSWER 1 OF 1  HCAPLUS  COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER:         2004:1003474  HCAPLUS
DOCUMENT NUMBER:          142:168720
TITLE:                    Canstatin, a endogenous inhibitor of
                          angiogenesis and tumor growth
AUTHOR(S):                Su, Ying; Zhu, Jian-si
CORPORATE SOURCE:         Institute of Cancer Research, Nan Hua University,
                          Hengyang, 421001, Peop. Rep. China
SOURCE:                   Chinese Journal of Cancer Research (2004), 16(3),
                          229-234
                          CODEN: CJCRFH; ISSN: 1000-9604
PUBLISHER:                Chinese Journal of Cancer Research
DOCUMENT TYPE:            Journal; General Review
LANGUAGE:                 English
AB    A review.  Canstatin is a novel inhibitor of angiogenesis and
      tumor growth, derived from the C-terminal globular non-collageneous (NCI)
      domain of the α2 chain of type IV collagen.  It inhibits endothelial
      cell proliferation and migration in a dose-dependent manner, and induces
      endothelial cell apoptosis.  In vivo expts. show that canstatin
      significantly inhibits solid tumor growth.  The canstatin mediated
      inhibition of tumor is related to apoptosis.  Canstatin- induced apoptosis
      is associated with phosphatidylinositol 3-kinase/Akt
      inhibition and is dependent upon signaling events transduced
      trough membrane death receptor.
REFERENCE COUNT:          20    THERE ARE 20 CITED REFERENCES AVAILABLE FOR THIS
                                RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT


=> d his

     (FILE 'HOME' ENTERED AT 17:51:52 ON 02 OCT 2006)

     FILE 'REGISTRY' ENTERED AT 17:52:01 ON 02 OCT 2006

     FILE 'HCAPLUS' ENTERED AT 17:54:03 ON 02 OCT 2006
```

Updated Search

10554187

```
L1        11157 S AKT OR ?AKT () ONCOGENE? () PROTEIN? OR AKT () KINASE () TRAN
L2          383 S L1 () INHIBIT?
L3           17 S L2 AND REVIEW/DT
L4            8 S L3 AND CANCER
L5            9 S L3 NOT L4
L6            0 S L5 AND HYPERINSULINISUM?
L7            1 S L4 AND INSULIN?
L8            7 S L4 NOT L7
L9            9 S L5 NOT L7
L10           0 S L9 AND HYPERPROLIFERATIVE () DISORDER?
L11           1 S L9 AND ANGIOGENESIS?

=> s l9 not l11
L12           8 L9 NOT L11

=> s l12 and restenosis?
          7776 RESTENOSIS?
L13           0 L12 AND RESTENOSIS?

=> s l12 and inflammation?
        159606 INFLAMMATION?
L14           0 L12 AND INFLAMMATION?

=> s l12 and autoimmune?
         48030 AUTOIMMUNE?
L15           0 L12 AND AUTOIMMUNE?

=> s l12 and allergy?
         44675 ALLERGY?
L16           0 L12 AND ALLERGY?

=> s l12 and asthma?
         33692 ASTHMA?
L17           0 L12 AND ASTHMA?

=> d l12, ibib abs hitstr, 1-8

L12  ANSWER 1 OF 8  HCAPLUS  COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER:         2006:488600  HCAPLUS
TITLE:                    Phosphoinositide 3-kinase/Akt signaling pathway and
                          its therapeutical implications for human acute myeloid
                          leukemia
AUTHOR(S):                Martelli, A. M.; Nyakern, M.; Tabellini, G.; Bortul,
                          R.; Tazzari, P. L.; Evangelisti, C.; Cocco, L.
CORPORATE SOURCE:         Cell Signalling Laboratory, Dipartimento di Scienze
                          Anatomiche Umane e Fisiopatologia dell'Apparato
                          Locomotore, Sezione di Anatomia Umana, Universita di
                          Bologna, Bologna, Italy
SOURCE:                   Leukemia (2006), 20(6), 911-928
                          CODEN: LEUKED; ISSN: 0887-6924
PUBLISHER:                Nature Publishing Group
DOCUMENT TYPE:            Journal; General Review
LANGUAGE:                 English
AB   The phosphoinositide 3-kinase (PI3K)/Akt signaling pathway is crucial to
     many aspects of cell growth, survival and apoptosis, and its constitutive
     activation has been implicated in the both the pathogenesis and the
     progression of a wide variety of neoplasias.  Hence, this pathway is an
     attractive target for the development of novel anticancer strategies.
     Recent studies showed that PI3K/Akt signaling is frequently activated in
```

acute myeloid leukemia (AML) patient blasts and strongly contributes to proliferation, survival and drug resistance of these cells. Upregulation of the PI3K/Akt network in AML may be due to several reasons, including FLT3, Ras or c-Kit mutations. Small mols. designed to selectively target key components of this signal transduction cascade induce apoptosis and/or markedly increase conventional drug sensitivity of AML blasts in vitro. Thus, inhibitory mols. are currently being developed for clin. use either as single agents or in combination with conventional therapies. However, the PI3K/Akt pathway is important for many physiol. cellular functions and, in particular, for insulin signaling, so that its blockade in vivo might cause severe systemic side effects. In this review, we summarize the existing knowledge about PI3K/Akt signaling in AML cells and we examine the rationale for targeting this fundamental signal transduction network by means of selective pharmacol. inhibitors.

REFERENCE COUNT: 250 THERE ARE 250 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 2 OF 8 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2005:1234702 HCAPLUS
DOCUMENT NUMBER: 145:116355
TITLE: Deguelin as a chemopreventive agent in mouse lung tumorigenesis induced by tobacco smoke carcinogens
AUTHOR(S): Hecht, Stephen S.
CORPORATE SOURCE: The Cancer Center, University of Minnesota, Minn., MN, 55455, USA
SOURCE: Journal of the National Cancer Institute (2005), 97(22), 1634-1635
CODEN: JNCIEQ; ISSN: 0027-8874
PUBLISHER: Oxford University Press
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English

AB A review. The research of Lee et al. (2005) entitled "Chemopreventive effects of deguelin, a novel Akt inhibitor, on tobacco-induced lung tumorigenesis" is reviewed with commentary and refs. The study by these authors showed that in mouse models, deguelin decreased the expression of pAkt in lungs and inhibited lung tumorigenesis induced by the tobacco smoke carcinogen benzo[a]pyrene (BAP) and 4(-methylnitrosamino)-1-(3-pyridyl)-1-butanone (NKK). The effects were particularly striking considering the relatively low dose of deguelin (4 mg/kg, twice a day) used in the chemoprevention study. Deguelin was effective when administered at the same time as BAP plus NKK or when given after carcinogen administration. An innovative aspect of this study was the use of microcomputed tomog. image anal. to detect lung tumors in live mice.

REFERENCE COUNT: 28 THERE ARE 28 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 3 OF 8 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2005:1068675 HCAPLUS
DOCUMENT NUMBER: 144:120596
TITLE: Accelerating lead development by microwave-enhanced medicinal chemistry
AUTHOR(S): Shipe, William D.; Wolkenberg, Scott E.; Lindsley, Craig W.
CORPORATE SOURCE: Department of Medicinal Chemistry, Merck Research Laboratories, West Point, PA, 19486, USA
SOURCE: Drug Discovery Today: Technologies (2005), 2(2), 155-161

Updated Search

10554187

CODEN: DDTTB5; ISSN: 1740-6749
URL: http://www.sciencedirect.com/science/journal/1740 6749
PUBLISHER: Elsevier B.V.
DOCUMENT TYPE: Journal; General Review; (online computer file)
LANGUAGE: English
AB A review. Microwave-assisted organic synthesis (MAOS) addresses the need for accelerated chemical synthesis by providing many advantages over classical thermal conditions. Microwave instruments produced by Biotage, CEM and Milestone enable chemical to be safely and reproducibly performed on various scales and in a parallel fashion. To illustrate the high utility of this technol. for lead development, our Akt kinase program will be described wherein MAOS played a pivotal role in the identification of isoenzyme-selective Akt inhibitors.
REFERENCE COUNT: 29 THERE ARE 29 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 4 OF 8 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2005:329631 HCAPLUS
DOCUMENT NUMBER: 142:456149
TITLE: Inhibitors of β-amyloid-induced toxicity by modulating the Akt signaling pathway
AUTHOR(S): Nakagami, Yasuhiro
CORPORATE SOURCE: Biological Research Laboratories, Sankyo Co., Ltd., Tokyo, 140-8710, Japan
SOURCE: Drug News & Perspectives (2004), 17(10), 655-660
CODEN: DNPEED; ISSN: 0214-0934
PUBLISHER: Prous Science
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
AB A review. The Akt signaling pathway plays a crucial role in neuronal survival, leading to inhibition of apoptosis. Many stimulants including neurotrophins are reported to activate this pathway in preclin. studies; however, there are no drugs for neurodegenerative diseases adopting such a concept on the market so far. Among neurodegenerative diseases, Alzheimer's disease is the most common and characterized by senile plaques and neurofibrillary tangles, which consist of β-amyloid and hyperphosphorylated tau, resp. Recent studies suggest that activation of Akt inhibits toxicity of β-amyloid and formation of neurofibrillary tangles, leading to protection of neurons against apoptosis. This review discusses the possibility of treatment of Alzheimer's disease by activating the Akt signaling pathway.
REFERENCE COUNT: 59 THERE ARE 59 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 5 OF 8 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2004:475523 HCAPLUS
DOCUMENT NUMBER: 142:195821
TITLE: The molecular mechanisms regulating the phosphorylation of the NADPH oxidase component p47phox by phosphoinositide 3-kinase
AUTHOR(S): Yamamori, Tohru; Inanami, Osamu; Nagahata, Hajime; Kuwabara, Mikinori
CORPORATE SOURCE: Lab. Radiat. Biol., Dep. Environ. Veterinary Sci., Grad. Sch. Veterinary Med., Hokkaido Univ., Sapporo, 060-0818, Japan
SOURCE: Jui Seikagaku (2003), 40(2), 63-76
CODEN: JSUEBY; ISSN: 1345-921X

Updated Search

10554187

PUBLISHER: Jui Seikagakkai
DOCUMENT TYPE: Journal; General Review
LANGUAGE: Japanese
AB A review. Superoxide production by NADPH oxidase is essential for the bactericidal properties of phagocytes. Phosphorylation of p47phox, one of the cytosolic components of NADPH oxidase, is a crucial step of the oxidase activation. Some evidences suggest that phosphoinositide 3-kinase (PI3K) is involved in p47phox phosphorylation, but it has not been fully understood how PI3K regulates it. The aim of this study was to examine the mechanism underlying the PI3K-regulation of p47phox phosphorylation. Pharmacol. inhibition of PI3K attenuated both fMLP-stimulated p47phox phosphorylation and NADPH oxidase activity in HL-60 cells differentiated to a neutrophil-like phenotype. Although fMLP elicited Akt activation in a PI3K-dependent manner, an Akt inhibitor had no effect on the oxidase activity triggered by fMLP. In vitro kinase assay revealed that Akt was unable to catalyze p47phox phosphorylation. Interestingly, the activation of cPKC and PKCδ after fMLP stimulation was dependent on PI3K. Furthermore, PI3K inhibitors reduced the activation of phospholipase Cγ2 without affecting tyrosine phosphorylation on it. These results suggest that PI3K regulates the phosphorylation of NADPH oxidase component p47phox by controlling diacylglycerol-dependent PKCs but not Akt.

L12 ANSWER 6 OF 8 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2004:361502 HCAPLUS
DOCUMENT NUMBER: 141:306789
TITLE: Anti apoptotic proteins as targets of chemotherapy
AUTHOR(S): Smitha, V. B.; Ruby, John Anto
CORPORATE SOURCE: Division of Cancer Biology, Rajiv Gandhi Centre for Biotechnology, Thiruvananthapuram, 695014, India
SOURCE: Amala Research Bulletin (2003), 23, 1-6
CODEN: ARBMCS; ISSN: 0971-4987
PUBLISHER: Amala Cancer Research Centre
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
AB A review focuses on different anti-apoptotic mols. over-expressed by various tumors and their role in regulating the effectiveness of antitumor chemotherapy. It describes NF-κB; Bcl-2; Akt; inhibitor of apoptosis; and heat shock protein.
REFERENCE COUNT: 45 THERE ARE 45 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 7 OF 8 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2003:453335 HCAPLUS
DOCUMENT NUMBER: 139:225909
TITLE: Decisions on life and death: FOXO forkhead transcription factors are in command when PKB/Akt is off duty
AUTHOR(S): Burgering, Boudewijn M. T.; Medema, Rene H.
CORPORATE SOURCE: Department of Physiological Chemistry and Center for Biomedical Genetics, University Medical Center Utrecht, Neth.
SOURCE: Journal of Leukocyte Biology (2003), 73(6), 689-701
CODEN: JLBIE7; ISSN: 0741-5400
PUBLISHER: Federation of American Societies for Experimental Biology
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
AB A review. Forkhead transcription factors of the FOXO family are important

Updated Search

downstream targets of protein kinase B (PKB)/Akt, a kinase shown to play a decisive role in cell proliferation and cell survival. Direct phosphorylation by PKB/Akt inhibits transcriptional activation by FOXO factors, causing their displacement from the nucleus into the cytoplasm. Work from recent years has shown that this family of transcription factors regulates the expression of a number of genes that are crucial for the proliferative status of a cell, as well as a number of genes involved in programmed cell death. As such, these transcription factors appear to play an essential role in many of the effects of PKB/Akt on cell proliferation and survival. Indeed, in cells of the hematopoietic system, mere activation of a FOXO factor is sufficient to activate a variety of proapoptotic genes and to trigger apoptosis. In contrast, in most other cell types, activation of FOXO blocks cellular proliferation and drives cells into a quiescent state. In such cell types, FOXO factors also provide the protective mechanisms that are requires to adapt to the altered metabolic state of quiescent cells. Thus, as PKB/Akt signaling is switched off, FOXO factors take over to determine the fate of a cell, long-term survival in a quiescent state, or programmed cell death. This review summarizes our current understanding of the mechanisms by which PKB/Akt and FOXO factors regulate these decisions.

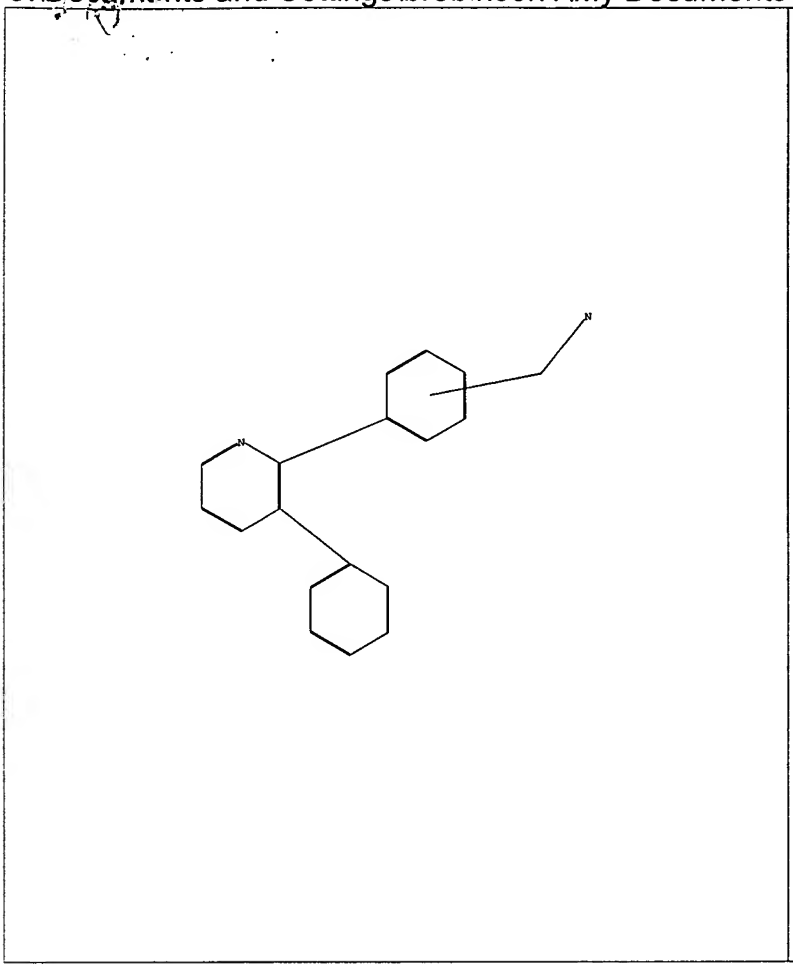
REFERENCE COUNT: 101 THERE ARE 101 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

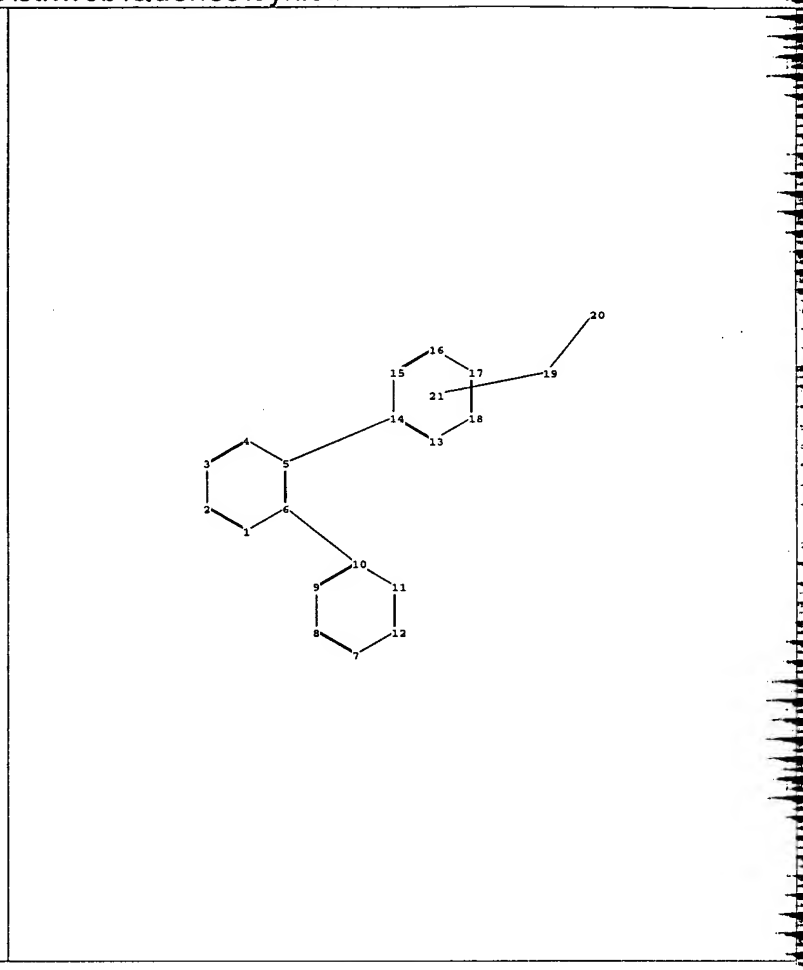
L12 ANSWER 8 OF 8 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2003:446535 HCAPLUS
DOCUMENT NUMBER: 139:63860
TITLE: Akt inhibits DNA damage by suppressing p73, p53, Forkhead or all three?
AUTHOR(S): Basu, Subham
CORPORATE SOURCE: Signal Transduction Lab., Cancer Res., UK
SOURCE: Cell Cycle (2003), 2(2), 69-70
CODEN: CCEYAS; ISSN: 1538-4101
PUBLISHER: Landes Bioscience
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English

AB A review discusses Akt regulation of DNA damage by suppression of transcription through p73, p53 and Forkhead. Recent data suggest that Akt regulation of p73-dependent DNA-damage is mediated primarily through YAP (Yes-associated protein), while Akt regulation of p53-dependent cell death is mediated through MDM2. Depending on the nature of the pro-apoptotic stimuli, DNA damaging or otherwise, the various Akt targets are possibly not only differentially regulated, as demonstrated by p53 and p73, but the same target may signal contrary effects, as seems to be the case for Forkhead. In an actual in vivo scenario, such as in a transformed cell that has evaded apoptosis by increasing Akt activity, the overall picture is probably generated by the coordinated regulation of a number of substrates, including but not limited to p53, p73 and Forkhead transcription factors.

REFERENCE COUNT: 9 THERE ARE 9 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

C:\Documents and Settings\brobinson1\My Documents\stnweb\Queries\6yk.str





chain nodes :
19 20
ring nodes :
1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18
chain bonds :
5-14 6-10 19-20
ring bonds :
1-2 1-6 2-3 3-4 4-5 5-6 7-8 7-12 8-9 9-10 10-11 11-12 13-14 13-18 14-15 15-16 16-17 17-18
exact/norm bonds :
19-20
exact bonds :
5-14 6-10
normalized bonds :
1-2 1-6 2-3 3-4 4-5 5-6 7-8 7-12 8-9 9-10 10-11 11-12 13-14 13-18 14-15 15-16 16-17 17-18
isolated ring systems :
containing 1 : 7 : 13 :

Match level :
1:Atom 2:Atom 3:Atom 4:Atom 5:Atom 6:Atom 7:Atom 8:Atom 9:Atom 10:Atom 11:Atom 12:Atom 13:Atom 14:Atom 15:Atom 16:Atom 17:Atom 18:Atom 19:CLASS 20:CLASS 21:Atom

10554187

Connecting via Winsock to STN

Welcome to STN International! Enter x:x

LOGINID:ssspta1612bxr

PASSWORD:
TERMINAL (ENTER 1, 2, 3, OR ?):2

* * * * * * * * * * Welcome to STN International * * * * * * * * * *

NEWS 1		Web Page URLs for STN Seminar Schedule - N. America
NEWS 2		"Ask CAS" for self-help around the clock
NEWS 3	FEB 27	New STN AnaVist pricing effective March 1, 2006
NEWS 4	MAY 10	CA/CAplus enhanced with 1900-1906 U.S. patent records
NEWS 5	MAY 11	KOREAPAT updates resume
NEWS 6	MAY 19	Derwent World Patents Index to be reloaded and enhanced
NEWS 7	MAY 30	IPC 8 Rolled-up Core codes added to CA/CAplus and USPATFULL/USPAT2
NEWS 8	MAY 30	The F-Term thesaurus is now available in CA/CAplus
NEWS 9	JUN 02	The first reclassification of IPC codes now complete in INPADOC
NEWS 10	JUN 26	TULSA/TULSA2 reloaded and enhanced with new search and and display fields
NEWS 11	JUN 28	Price changes in full-text patent databases EPFULL and PCTFULL
NEWS 12	JUl 11	CHEMSAFE reloaded and enhanced
NEWS 13	JUl 14	FSTA enhanced with Japanese patents
NEWS 14	JUl 19	Coverage of Research Disclosure reinstated in DWPI
NEWS 15	AUG 09	INSPEC enhanced with 1898-1968 archive
NEWS 16	AUG 28	ADISCTI Reloaded and Enhanced
NEWS 17	AUG 30	CA(SM)/CAplus(SM) Austrian patent law changes
NEWS 18	SEP 11	CA/CAplus enhanced with more pre-1907 records
NEWS 19	SEP 21	CA/CAplus fields enhanced with simultaneous left and right truncation
NEWS 20	SEP 25	CA(SM)/CAplus(SM) display of CA Lexicon enhanced
NEWS 21	SEP 25	CAS REGISTRY(SM) no longer includes Concord 3D coordinates
NEWS 22	SEP 25	CAS REGISTRY(SM) updated with amino acid codes for pyrrolysine
NEWS 23	SEP 28	CEABA-VTB classification code fields reloaded with new classification scheme
NEWS 24	OCT 02	MARPAT(R) now updated daily

NEWS EXPRESS JUNE 30 CURRENT WINDOWS VERSION IS V8.01b, CURRENT MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP), AND CURRENT DISCOVER FILE IS DATED 26 JUNE 2006.

NEWS HOURS	STN Operating Hours Plus Help Desk Availability
NEWS LOGIN	Welcome Banner and News Items
NEWS IPC8	For general information regarding STN implementation of IPC 8
NEWS X25	X.25 communication option no longer available

Enter NEWS followed by the item number or name to see news on that specific topic.

All use of STN is subject to the provisions of the STN Customer

Updated Search

10554187

agreement. Please note that this agreement limits use to scientific research. Use for software development or design or implementation of commercial gateways or other similar uses is prohibited and may result in loss of user privileges and other penalties.

* * * * * * * * * * * * * * * * STN Columbus * * * * * * * * * * * * * * * * *

FILE 'HOME' ENTERED AT 14:36:42 ON 02 OCT 2006

=> file reg

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	0.84	0.84

FILE 'REGISTRY' ENTERED AT 14:39:04 ON 02 OCT 2006
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.
PLEASE SEE "HELP USAGETERMS" FOR DETAILS.
COPYRIGHT (C) 2006 American Chemical Society (ACS)

Property values tagged with IC are from the ZIC/VINITI data file provided by InfoChem.

STRUCTURE FILE UPDATES: 29 SEP 2006 HIGHEST RN 909185-74-6
DICTIONARY FILE UPDATES: 29 SEP 2006 HIGHEST RN 909185-74-6

New CAS Information Use Policies, enter HELP USAGETERMS for details.

TSCA INFORMATION NOW CURRENT THROUGH June 30, 2006

Please note that search-term pricing does apply when conducting SmartSELECT searches.

REGISTRY includes numerically searchable data for experimental and predicted properties as well as tags indicating availability of experimental property data in the original document. For information on property searching in REGISTRY, refer to:

http://www.cas.org/ONLINE/UG/regprops.html

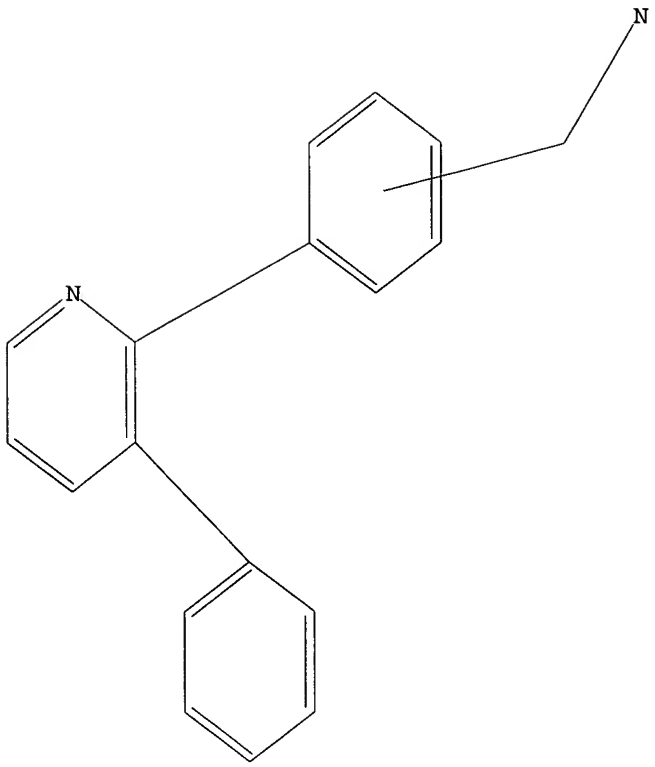
=>
Uploading C:\Documents and Settings\brobinson1\My Documents\stnweb\Queries\6yk.str

L1 STRUCTURE UPLOADED

=> d l1
L1 HAS NO ANSWERS
L1 STR

Updated Search

10554187



Structure attributes must be viewed using STN Express query preparation.

```
=> s l1
SAMPLE SEARCH INITIATED 14:41:01 FILE 'REGISTRY'
SAMPLE SCREEN SEARCH COMPLETED -      402 TO ITERATE

100.0% PROCESSED      402 ITERATIONS                                 1 ANSWERS
SEARCH TIME: 00.00.01

FULL FILE PROJECTIONS:  ONLINE  **COMPLETE**
                        BATCH   **COMPLETE**
PROJECTED ITERATIONS:         6838 TO     9242
PROJECTED ANSWERS:               1 TO       80

L2             1 SEA SSS SAM L1

=> s l1 full
THE ESTIMATED SEARCH COST FOR FILE 'REGISTRY' IS 166.50 U.S. DOLLARS
DO YOU WANT TO CONTINUE WITH THIS REQUEST? (Y)/N or END:y
FULL SEARCH INITIATED 14:41:06 FILE 'REGISTRY'
FULL SCREEN SEARCH COMPLETED -     8595 TO ITERATE

100.0% PROCESSED     8595 ITERATIONS                                36 ANSWERS
SEARCH TIME: 00.00.01

L3            36 SEA SSS FUL L1

=> file hcaplus
```

Updated Search

10554187

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	168.26	169.10

FILE 'HCAPLUS' ENTERED AT 14:41:10 ON 02 OCT 2006
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.
PLEASE SEE "HELP USAGETERMS" FOR DETAILS.

FILE COVERS 1907 - 2 Oct 2006 VOL 145 ISS 15
FILE LAST UPDATED: 1 Oct 2006 (20061001/ED)

New CAS Information Use Policies, enter HELP USAGETERMS for details.

This file contains CAS Registry Numbers for easy and accurate substance identification.

```
=> s l3
L4            4 L3

=> s l3/thu
              4 L3
         817176 THU/RL
L5            4 L3/THU
                  (L3 (L) THU/RL)

=> s l5 and duggan, m?/au
            284 DUGGAN, M?/AU
L6            2 L5 AND DUGGAN, M?/AU

=> d l6, ibib abs hitstr, 1-2
```

L6 ANSWER 1 OF 2 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER:	2005:86368 HCAPLUS
DOCUMENT NUMBER:	142:211437
TITLE:	Discovery of 2,3,5-trisubstituted pyridine derivatives as potent Akt1 and Akt2 dual inhibitors
AUTHOR(S):	Zhao, Zhijian; Leister, William H.; Robinson, Ronald G.; Barnett, Stanley F.; Defeo-Jones, Deborah; Jones, Raymond E.; Hartman, George D.; Huff, Joel R.; Huber, Hans E.; Duggan, Mark E.; Lindsley, Craig W.
CORPORATE SOURCE:	Department of Medicinal Chemistry, Technology Enabled Synthesis Group, Merck Research Laboratories, Merck & Co., West Point, PA, 19486, USA
SOURCE:	Bioorganic & Medicinal Chemistry Letters (2005), 15(4), 905-909 CODEN: BMCLE8; ISSN: 0960-894X
PUBLISHER:	Elsevier B.V.

Updated Search

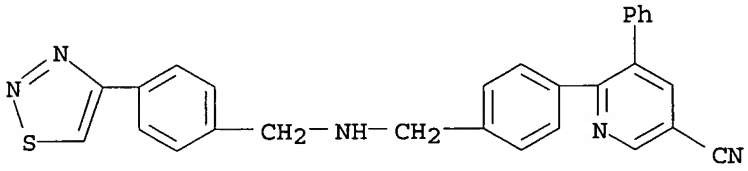
10554187

DOCUMENT TYPE: Journal
LANGUAGE: English
OTHER SOURCE(S): CASREACT 142:211437

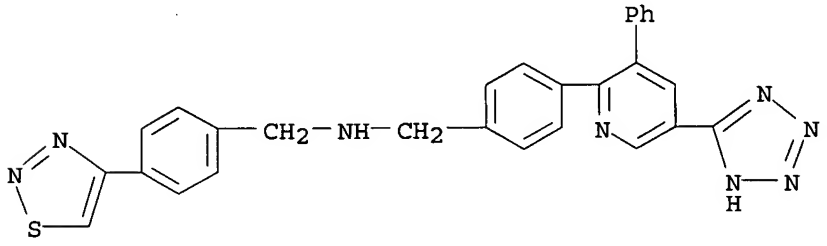
AB This letter describes the discovery of a novel series of dual Akt1/Akt2 kinase inhibitors, based on a 2,3,5-trisubstituted pyridine scaffold. Compds. from this series, which contain a 5-tetrazolyl moiety, exhibit more potent inhibition of Akt2 than Akt1.

IT 790659-59-5P 790659-68-6P
RL: PAC (Pharmacological activity); PKT (Pharmacokinetics); SPN (Synthetic preparation); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(preparation of 2,3,5-trisubstituted pyridine derivs. as potent Akt1/Akt2 dual inhibitors)

RN 790659-59-5 HCAPLUS
CN 3-Pyridinecarbonitrile, 5-phenyl-6-[4-[[[[4-(1,2,3-thiadiazol-4-yl)phenyl]methyl]amino]methyl]phenyl]- (9CI) (CA INDEX NAME)



RN 790659-68-6 HCAPLUS
CN Benzenemethanamine, N-[[4-[3-phenyl-5-(1H-tetrazol-5-yl)-2-pyridinyl]phenyl]methyl]-4-(1,2,3-thiadiazol-4-yl)- (9CI) (CA INDEX NAME)



REFERENCE COUNT: 18 THERE ARE 18 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 2 OF 2 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2004:964999 HCAPLUS
DOCUMENT NUMBER: 141:406038
TITLE: Substituted pyridine compounds as inhibitors of protein kinase Akt activity for treating cancer
INVENTOR(S): Duggan, Mark E.; Lindsley, Craig W.; Wu, Zhicai; Zhao, Zhijian; Hartnett, John C.
PATENT ASSIGNEE(S): Merck & Co., Inc., USA
SOURCE: PCT Int. Appl., 58 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

Updated Search

10554187

PATENT NO.	KIND	DATE	APPLICATION NO.		DATE
WO 2004096135	A2	20041111	WO 2004-US12265		20040420
WO 2004096135	A3	20050324			
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW					
RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG					
AU 2004233835	A1	20041111	AU 2004-233835		20040420
CA 2522435	AA	20041111	CA 2004-2522435		20040420
EP 1631548	A2	20060308	EP 2004-750420		20040420
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, CY, TR, BG, CZ, EE, HU, PL, SK					
CN 1809536	A	20060726	CN 2004-80017036		20040420
PRIORITY APPLN. INFO.:			US 2003-465125P	P	20030424
			WO 2004-US12265	W	20040420

OTHER SOURCE(S): MARPAT 141:406038

AB The present invention is directed to compds. which contain a substituted pyridine moiety which inhibit the activity of Akt, a serine/threonine protein kinase. The invention is further directed to chemotherapeutic compns. containing the compds. of this invention and methods for treating cancer comprising administration of the compds. of the invention.

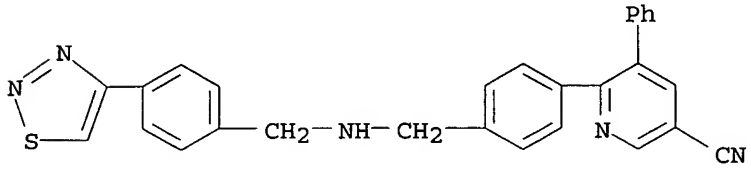
IT 790659-74-4P
RL: BSU (Biological study, unclassified); PAC (Pharmacological activity); RCT (Reactant); SPN (Synthetic preparation); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); RACT (Reactant or reagent); USES (Uses)
(substituted pyridine compds. as inhibitors of protein kinase Akt activity for treating cancer)

RN 790659-74-4 HCAPLUS

CN 3-Pyridinecarbonitrile, 5-phenyl-6-[4-[[[[4-(1,2,3-thiadiazol-4-yl)phenyl]methyl]amino]methyl]phenyl]-, trifluoroacetate (9CI) (CA INDEX NAME)

CM 1

CRN 790659-59-5
CMF C28 H21 N5 S

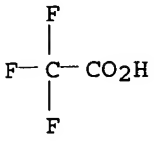


CM 2

Updated Search

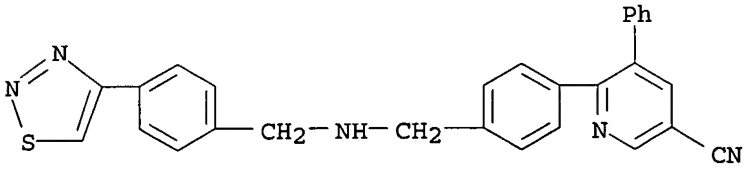
10554187

CRN 76-05-1
CMF C2 H F3 O2



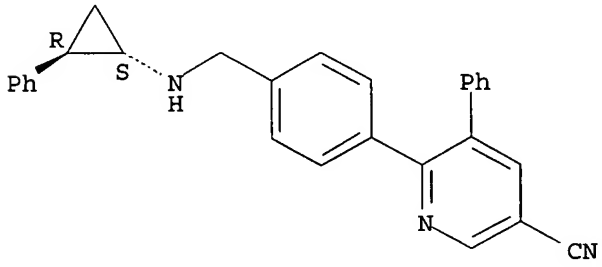
IT 790659-59-5P 790659-60-8P 790659-61-9P
790659-62-0P 790659-63-1P 790659-64-2P
790659-65-3P 790659-66-4P 790659-67-5P
790659-68-6P 790659-69-7P 790659-70-0P
790659-71-1P 790659-72-2P 790659-73-3P
790659-75-5P 790659-76-6P 790659-77-7P
790659-78-8P 790659-79-9P 790659-80-2P
790659-81-3P 790659-82-4P 790659-83-5P
790659-84-6P
RL: BSU (Biological study, unclassified); PAC (Pharmacological activity); SPN (Synthetic preparation); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(substituted pyridine compds. as inhibitors of protein kinase Akt activity for treating cancer)

RN 790659-59-5 HCAPLUS
CN 3-Pyridinecarbonitrile, 5-phenyl-6-[4-[[[[4-(1,2,3-thiadiazol-4-yl)phenyl]methyl]amino]methyl]phenyl]- (9CI) (CA INDEX NAME)



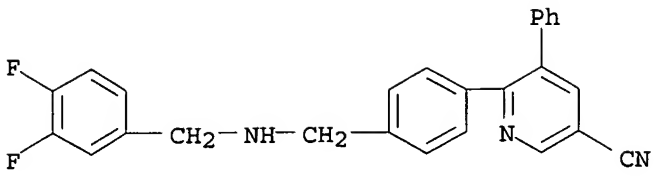
RN 790659-60-8 HCAPLUS
CN 3-Pyridinecarbonitrile, 5-phenyl-6-[4-[[[(1S,2R)-2-phenylcyclopropyl]amino]methyl]phenyl]- (9CI) (CA INDEX NAME)

Absolute stereochemistry.

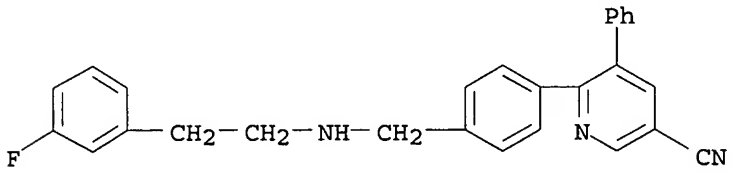


RN 790659-61-9 HCAPLUS
CN 3-Pyridinecarbonitrile, 6-[4-[[[(3,4-difluorophenyl)methyl]amino]methyl]phenyl]-5-phenyl- (9CI) (CA INDEX NAME)

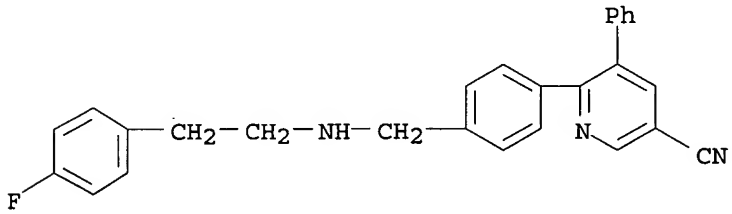
Updated Search



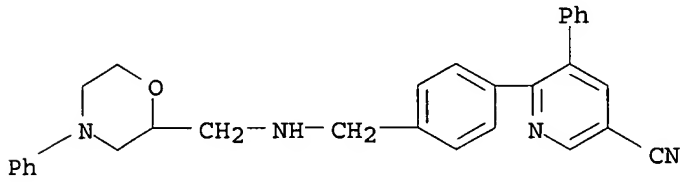
RN 790659-62-0 HCAPLUS
CN 3-Pyridinecarbonitrile, 6-[4-[[[2-(3-fluorophenyl)ethyl]amino]methyl]phenyl]-5-phenyl- (9CI) (CA INDEX NAME)



RN 790659-63-1 HCAPLUS
CN 3-Pyridinecarbonitrile, 6-[4-[[[2-(4-fluorophenyl)ethyl]amino]methyl]phenyl]-5-phenyl- (9CI) (CA INDEX NAME)

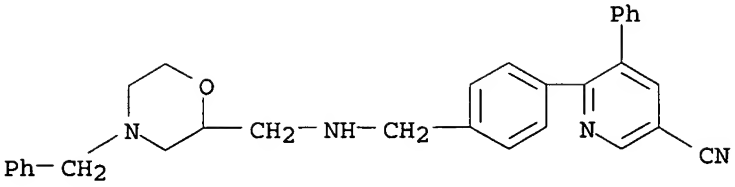


RN 790659-64-2 HCAPLUS
CN 3-Pyridinecarbonitrile, 5-phenyl-6-[4-[[[(4-phenyl-2-morpholinyl)methyl]amino]methyl]phenyl]- (9CI) (CA INDEX NAME)



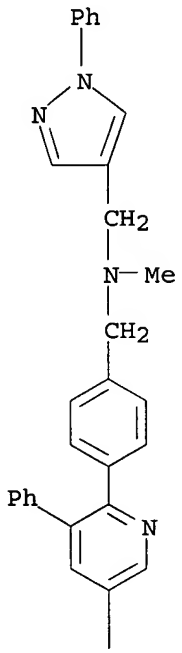
RN 790659-65-3 HCAPLUS
CN 3-Pyridinecarbonitrile, 5-phenyl-6-[4-[[[[4-(phenylmethyl)-2-morpholinyl]methyl]amino]methyl]phenyl]- (9CI) (CA INDEX NAME)

Updated Search



RN 790659-66-4 HCAPLUS
CN 3-Pyridinecarbonitrile, 6-[4-[[methyl[(1-phenyl-1H-pyrazol-4-yl)methyl]amino]methyl]phenyl]-5-phenyl- (9CI) (CA INDEX NAME)

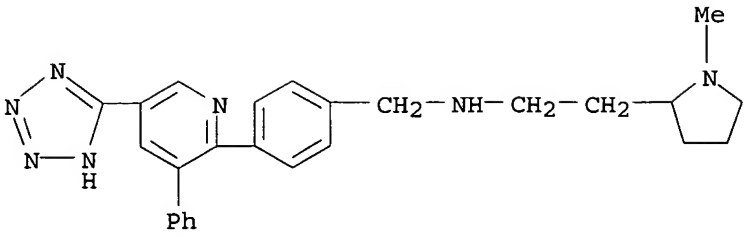
PAGE 1-A



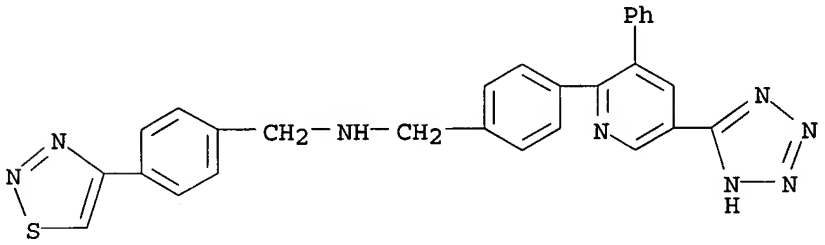
PAGE 2-A

RN 790659-67-5 HCAPLUS
CN 2-Pyrrolidineethanamine, 1-methyl-N-[[4-[3-phenyl-5-(1H-tetrazol-5-yl)-2-pyridinyl]phenyl]methyl]- (9CI) (CA INDEX NAME)

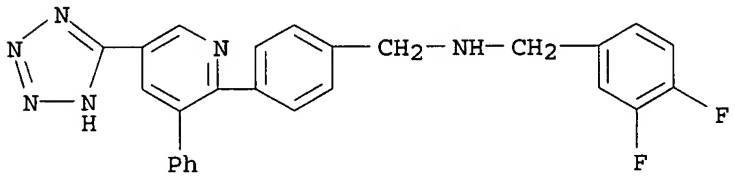
Updated Search



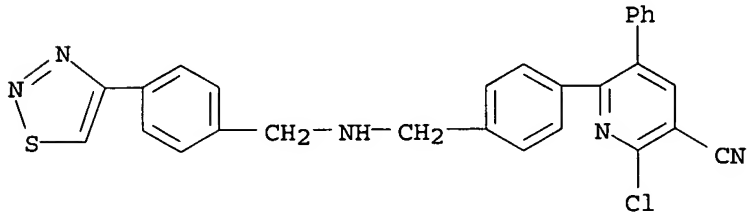
RN 790659-68-6 HCAPLUS
CN Benzenemethanamine, N-[[4-[3-phenyl-5-(1H-tetrazol-5-yl)-2-pyridinyl]phenyl]methyl]-4-(1,2,3-thiadiazol-4-yl)- (9CI) (CA INDEX NAME)



RN 790659-69-7 HCAPLUS
CN Benzenemethanamine, 3,4-difluoro-N-[[4-[3-phenyl-5-(1H-tetrazol-5-yl)-2-pyridinyl]phenyl]methyl]- (9CI) (CA INDEX NAME)

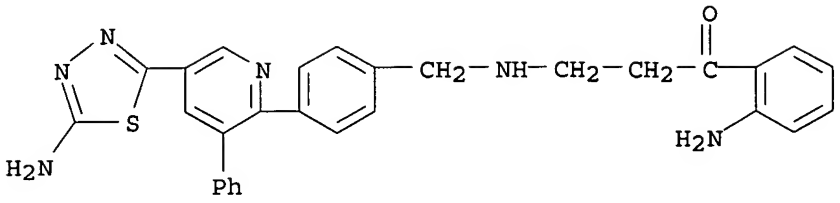


RN 790659-70-0 HCAPLUS
CN 3-Pyridinecarbonitrile, 2-chloro-5-phenyl-6-[4-[[[[4-(1,2,3-thiadiazol-4-yl)phenyl]methyl]amino]methyl]phenyl]- (9CI) (CA INDEX NAME)

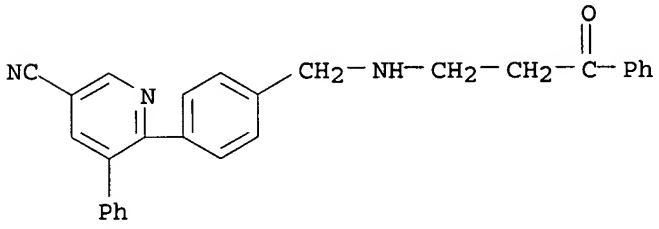


RN 790659-71-1 HCAPLUS
CN 1-Propanone, 1-(2-aminophenyl)-3-[[[4-[5-(5-amino-1,3,4-thiadiazol-2-yl)-3-phenyl-2-pyridinyl]phenyl]methyl]amino]- (9CI) (CA INDEX NAME)

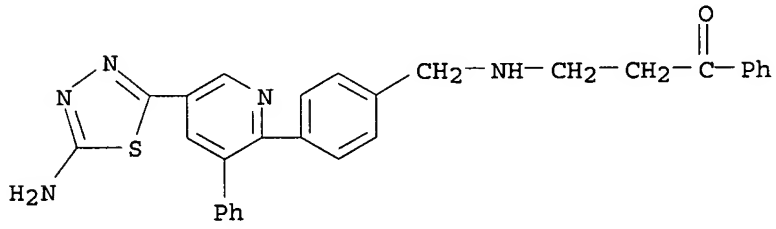
Updated Search



RN 790659-72-2 HCAPLUS
CN 3-Pyridinecarbonitrile, 6-[4-[[(3-oxo-3-phenylpropyl)amino]methyl]phenyl]-5-phenyl- (9CI) (CA INDEX NAME)



RN 790659-73-3 HCAPLUS
CN 1-Propanone, 3-[[[4-[5-(5-amino-1,3,4-thiadiazol-2-yl)-3-phenyl-2-pyridinyl]phenyl]methyl]amino]-1-phenyl- (9CI) (CA INDEX NAME)



RN 790659-75-5 HCAPLUS
CN 3-Pyridinecarbonitrile, 5-phenyl-6-[4-[[[(1S,2R)-2-phenylcyclopropyl]amino]methyl]phenyl]-, trifluoroacetate (9CI) (CA INDEX NAME)

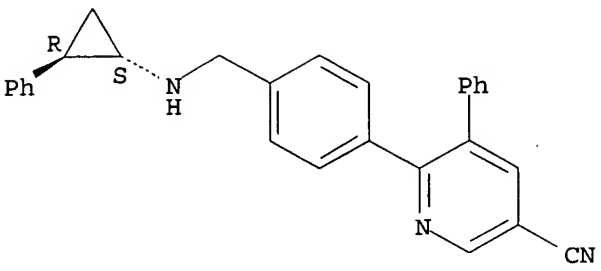
CM 1

CRN 790659-60-8
CMF C28 H23 N3

Absolute stereochemistry.

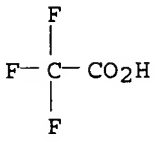
Updated Search

10554187



CM 2

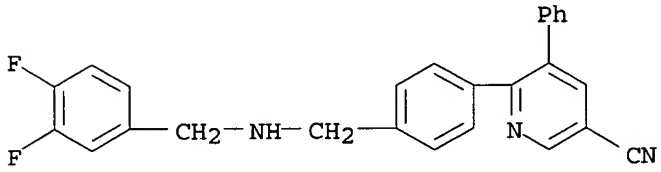
CRN 76-05-1
CMF C2 H F3 O2



RN 790659-76-6 HCAPLUS
CN 3-Pyridinecarbonitrile, 6-[4-[[[(3,4-difluorophenyl)methyl]amino]methyl]ph
enyl]-5-phenyl-, trifluoroacetate (9CI) (CA INDEX NAME)

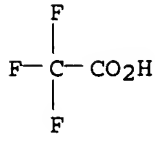
CM 1

CRN 790659-61-9
CMF C26 H19 F2 N3



CM 2

CRN 76-05-1
CMF C2 H F3 O2



RN 790659-77-7 HCAPLUS

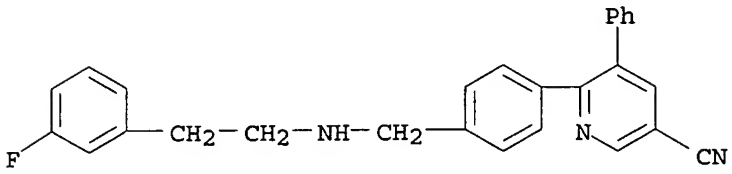
Updated Search

10554187

CN 3-Pyridinecarbonitrile, 6-[4-[[[2-(3-fluorophenyl)ethyl]amino]methyl]phenyl]-5-phenyl-, trifluoroacetate (9CI) (CA INDEX NAME)

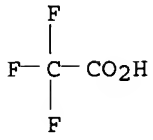
CM 1

CRN 790659-62-0
CMF C27 H22 F N3



CM 2

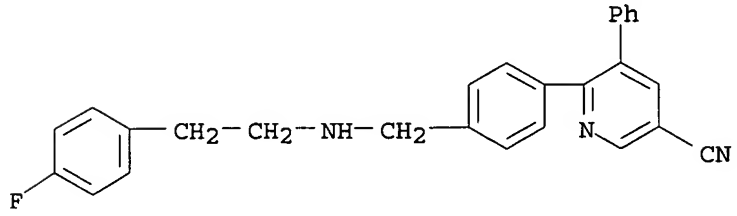
CRN 76-05-1
CMF C2 H F3 O2



RN 790659-78-8 HCAPLUS
CN 3-Pyridinecarbonitrile, 6-[4-[[[2-(4-fluorophenyl)ethyl]amino]methyl]phenyl]-5-phenyl-, trifluoroacetate (9CI) (CA INDEX NAME)

CM 1

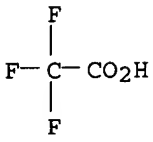
CRN 790659-63-1
CMF C27 H22 F N3



CM 2

CRN 76-05-1
CMF C2 H F3 O2

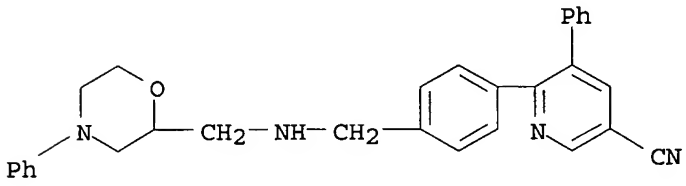
Updated Search



RN 790659-79-9 HCAPLUS
CN 3-Pyridinecarbonitrile, 5-phenyl-6-[4-[[[(4-phenyl-2-morpholinyl)methyl]amino]methyl]phenyl]-, trifluoroacetate (9CI) (CA INDEX NAME)

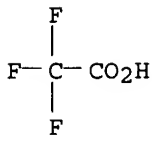
CM 1

CRN 790659-64-2
CMF C30 H28 N4 O



CM 2

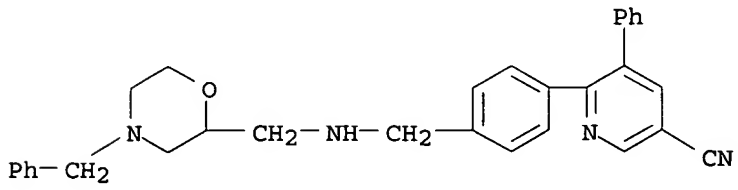
CRN 76-05-1
CMF C2 H F3 O2



RN 790659-80-2 HCAPLUS
CN 3-Pyridinecarbonitrile, 5-phenyl-6-[4-[[[[4-(phenylmethyl)-2-morpholinyl]methyl]amino]methyl]phenyl]-, trifluoroacetate (9CI) (CA INDEX NAME)

CM 1

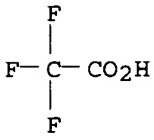
CRN 790659-65-3
CMF C31 H30 N4 O



Updated Search

CM 2

CRN 76-05-1
CMF C2 H F3 O2

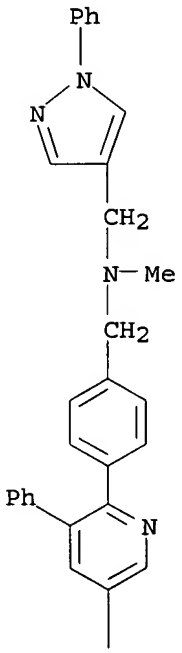


RN 790659-81-3 HCAPLUS
CN 3-Pyridinecarbonitrile, 6-[4-[[methyl[(1-phenyl-1H-pyrazol-4-yl)methyl]amino]methyl]phenyl]-5-phenyl-, trifluoroacetate (9CI) (CA INDEX NAME)

CM 1

CRN 790659-66-4
CMF C30 H25 N5

PAGE 1-A

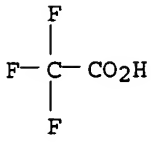


PAGE 2-A

Updated Search

CM 2

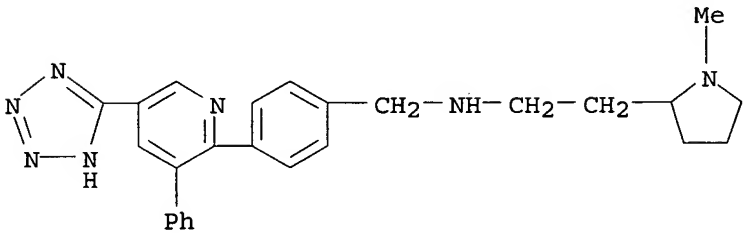
CRN 76-05-1
CMF C2 H F3 O2



RN 790659-82-4 HCAPLUS
CN 2-Pyrrolidineethanamine, 1-methyl-N-[[4-[3-phenyl-5-(1H-tetrazol-5-yl)-2-pyridinyl]phenyl]methyl]-, trifluoroacetate (9CI) (CA INDEX NAME)

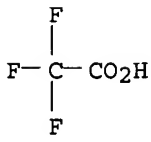
CM 1

CRN 790659-67-5
CMF C26 H29 N7



CM 2

CRN 76-05-1
CMF C2 H F3 O2

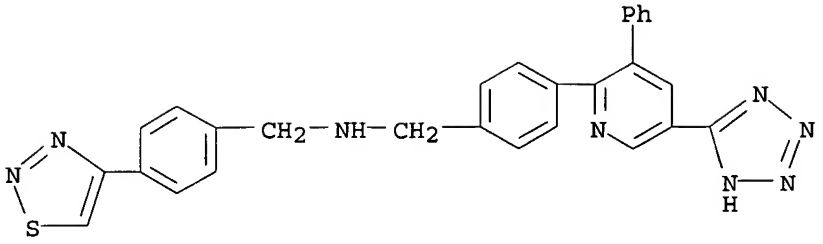


RN 790659-83-5 HCAPLUS
CN Benzenemethanamine, N-[[4-[3-phenyl-5-(1H-tetrazol-5-yl)-2-pyridinyl]phenyl]methyl]-4-(1,2,3-thiadiazol-4-yl)-, trifluoroacetate (9CI) (CA INDEX NAME)

CM 1

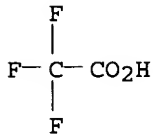
CRN 790659-68-6
CMF C28 H22 N8 S

Updated Search



CM 2

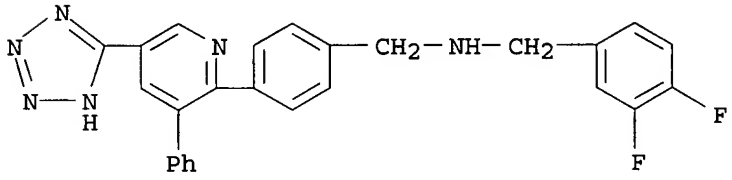
CRN 76-05-1
CMF C2 H F3 O2



RN 790659-84-6 HCAPLUS
CN Benzenemethanamine, 3,4-difluoro-N-[[4-[3-phenyl-5-(1H-tetrazol-5-yl)-2-pyridinyl]phenyl]methyl]-, trifluoroacetate (9CI) (CA INDEX NAME)

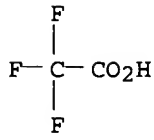
CM 1

CRN 790659-69-7
CMF C26 H20 F2 N6



CM 2

CRN 76-05-1
CMF C2 H F3 O2

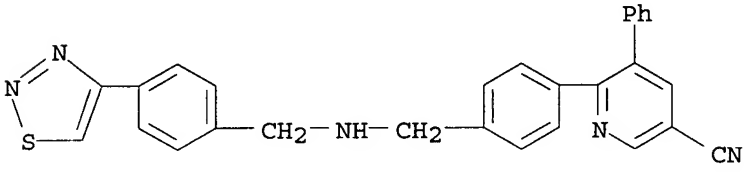


IT 790659-59-5D, salts, stereoisomers 790659-60-8D, salts, stereoisomers 790659-61-9D, salts, stereoisomers

Updated Search

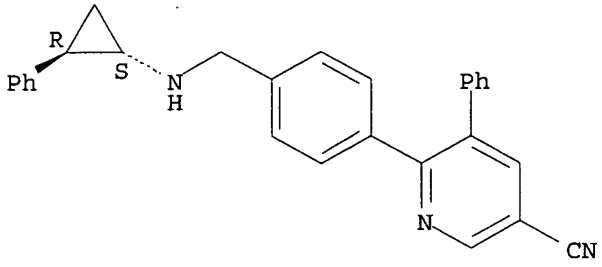
790659-62-0D, salts, stereoisomers 790659-63-1D, salts, stereoisomers 790659-64-2D, salts, stereoisomers 790659-65-3D, salts, stereoisomers 790659-66-4D, salts, stereoisomers 790659-67-5D, salts, stereoisomers 790659-68-6D, salts, stereoisomers 790659-69-7D, salts, stereoisomers 790659-70-0D, salts, stereoisomers 790659-71-1D, salts, stereoisomers 790659-72-2D, salts, stereoisomers 790659-73-3D, salts, stereoisomers 790659-85-7 790659-86-8
RL: BSU (Biological study, unclassified); PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(substituted pyridine compds. as inhibitors of protein kinase Akt activity for treating cancer)

RN 790659-59-5 HCAPLUS
CN 3-Pyridinecarbonitrile, 5-phenyl-6-[4-[[[[4-(1,2,3-thiadiazol-4-yl)phenyl]methyl]amino]methyl]phenyl]- (9CI) (CA INDEX NAME)

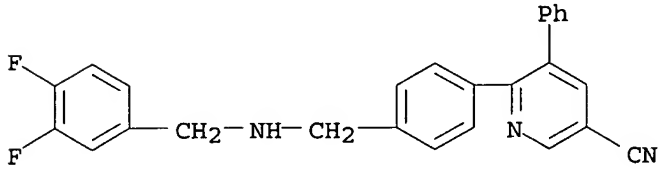


RN 790659-60-8 HCAPLUS
CN 3-Pyridinecarbonitrile, 5-phenyl-6-[4-[[[(1S,2R)-2-phenylcyclopropyl]amino]methyl]phenyl]- (9CI) (CA INDEX NAME)

Absolute stereochemistry.



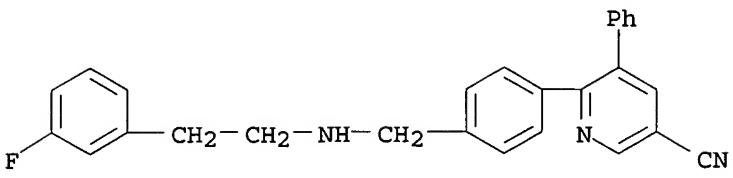
RN 790659-61-9 HCAPLUS
CN 3-Pyridinecarbonitrile, 6-[4-[[[(3,4-difluorophenyl)methyl]amino]methyl]phenyl]-5-phenyl- (9CI) (CA INDEX NAME)



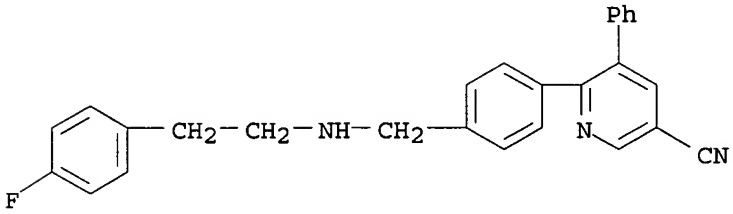
RN 790659-62-0 HCAPLUS
CN 3-Pyridinecarbonitrile, 6-[4-[[[2-(3-fluorophenyl)ethyl]amino]methyl]pheny

Updated Search

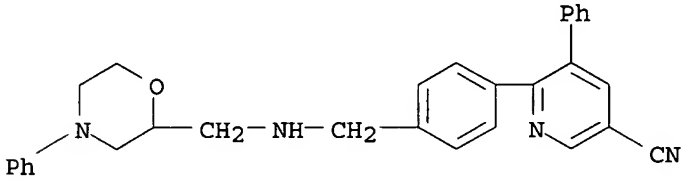
l]-5-phenyl- (9CI) (CA INDEX NAME)



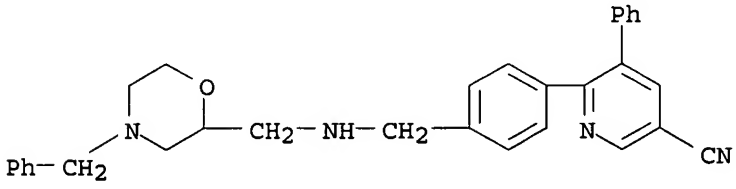
RN 790659-63-1 HCAPLUS
CN 3-Pyridinecarbonitrile, 6-[4-[[[2-(4-fluorophenyl)ethyl]amino]methyl]pheny
l]-5-phenyl- (9CI) (CA INDEX NAME)



RN 790659-64-2 HCAPLUS
CN 3-Pyridinecarbonitrile, 5-phenyl-6-[4-[[[(4-phenyl-2-
morpholinyl)methyl]amino]methyl]phenyl]- (9CI) (CA INDEX NAME)



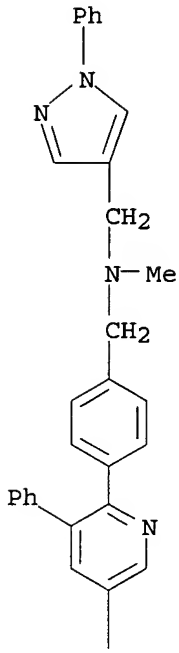
RN 790659-65-3 HCAPLUS
CN 3-Pyridinecarbonitrile, 5-phenyl-6-[4-[[[[4-(phenylmethyl)-2-
morpholinyl]methyl]amino]methyl]phenyl]- (9CI) (CA INDEX NAME)



RN 790659-66-4 HCAPLUS
CN 3-Pyridinecarbonitrile, 6-[4-[[methyl[(1-phenyl-1H-pyrazol-4-
yl)methyl]amino]methyl]phenyl]-5-phenyl- (9CI) (CA INDEX NAME)

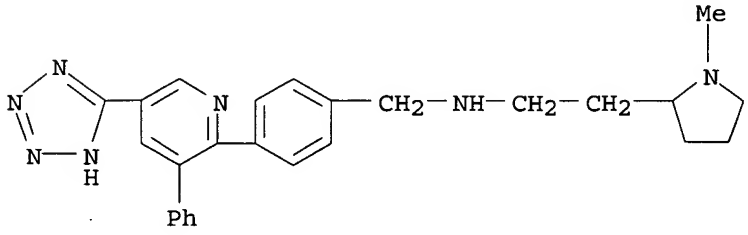
Updated Search

PAGE 1-A



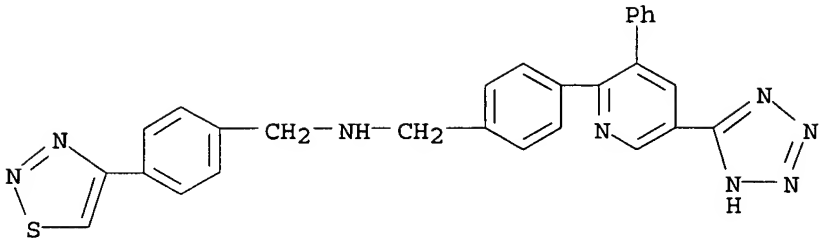
PAGE 2-A

RN 790659-67-5 HCAPLUS
CN 2-Pyrrolidineethanamine, 1-methyl-N-[[4-[3-phenyl-5-(1H-tetrazol-5-yl)-2-pyridinyl]phenyl]methyl]- (9CI) (CA INDEX NAME)

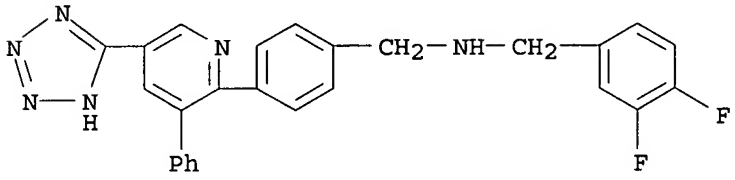


RN 790659-68-6 HCAPLUS
CN Benzenemethanamine, N-[[4-[3-phenyl-5-(1H-tetrazol-5-yl)-2-pyridinyl]phenyl]methyl]-4-(1,2,3-thiadiazol-4-yl)- (9CI) (CA INDEX NAME)

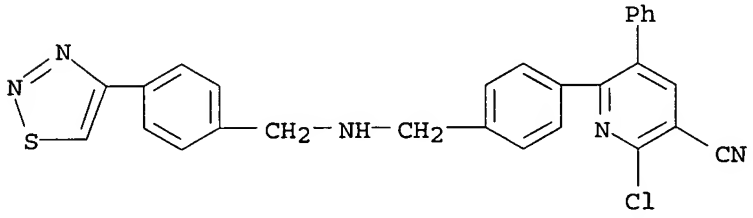
Updated Search



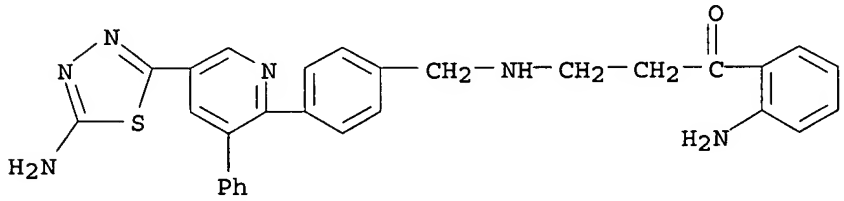
RN 790659-69-7 HCAPLUS
CN Benzenemethanamine, 3,4-difluoro-N-[[4-[3-phenyl-5-(1H-tetrazol-5-yl)-2-pyridinyl]phenyl]methyl]- (9CI) (CA INDEX NAME)



RN 790659-70-0 HCAPLUS
CN 3-Pyridinecarbonitrile, 2-chloro-5-phenyl-6-[4-[[[[4-(1,2,3-thiadiazol-4-yl)phenyl]methyl]amino]methyl]phenyl]- (9CI) (CA INDEX NAME)

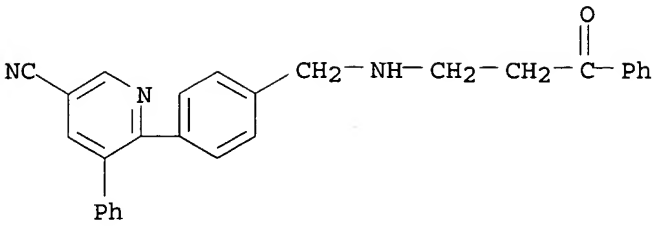


RN 790659-71-1 HCAPLUS
CN 1-Propanone, 1-(2-aminophenyl)-3-[[[4-[5-(5-amino-1,3,4-thiadiazol-2-yl)-3-phenyl-2-pyridinyl]phenyl]methyl]amino]- (9CI) (CA INDEX NAME)

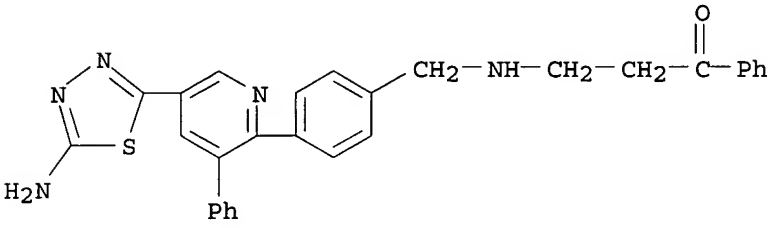


RN 790659-72-2 HCAPLUS
CN 3-Pyridinecarbonitrile, 6-[4-[[(3-oxo-3-phenylpropyl)amino]methyl]phenyl]-5-phenyl- (9CI) (CA INDEX NAME)

Updated Search



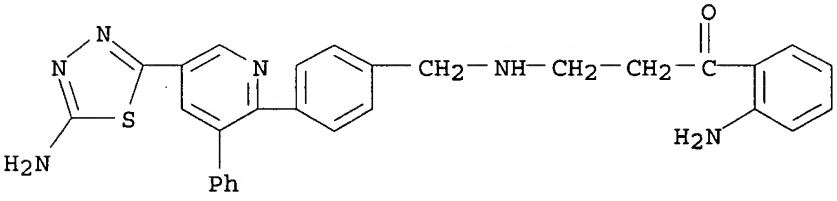
RN 790659-73-3 HCAPLUS
CN 1-Propanone, 3-[[[4-[5-(5-amino-1,3,4-thiadiazol-2-yl)-3-phenyl-2-pyridinyl]phenyl]methyl]amino]-1-phenyl- (9CI) (CA INDEX NAME)



RN 790659-85-7 HCAPLUS
CN 1-Propanone, 1-(2-aminophenyl)-3-[[[4-[5-(5-amino-1,3,4-thiadiazol-2-yl)-3-phenyl-2-pyridinyl]phenyl]methyl]amino]-, trifluoroacetate (9CI) (CA INDEX NAME)

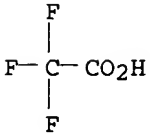
CM 1

CRN 790659-71-1
CMF C29 H26 N6 O S



CM 2

CRN 76-05-1
CMF C2 H F3 O2



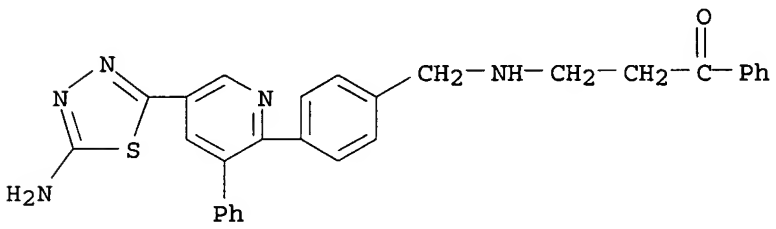
Updated Search

10554187

RN 790659-86-8 HCAPLUS
CN 1-Propanone, 3-[[[4-[5-(5-amino-1,3,4-thiadiazol-2-yl)-3-phenyl-2-pyridinyl]phenyl]methyl]amino]-1-phenyl-, trifluoroacetate (9CI) (CA INDEX NAME)

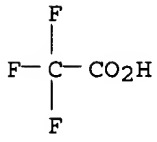
CM 1

CRN 790659-73-3
CMF C29 H25 N5 O S



CM 2

CRN 76-05-1
CMF C2 H F3 O2



=> d his

(FILE 'HOME' ENTERED AT 14:36:42 ON 02 OCT 2006)

FILE 'REGISTRY' ENTERED AT 14:39:04 ON 02 OCT 2006
L1 STRUCTURE UPLOADED
L2 1 S L1
L3 36 S L1 FULL

FILE 'HCAPLUS' ENTERED AT 14:41:10 ON 02 OCT 2006
L4 4 S L3
L5 4 S L3/THU
L6 2 S L5 AND DUGGAN, M?/AU

=> s l5 not l6
L7 2 L5 NOT L6

=> s l7 and lindsley, c?/au
100 LINDSLEY, C?/AU
L8 0 L7 AND LINDSLEY, C?/AU

=> s l7 and wu, z?/au

Updated Search

10554187

10714 WU, Z?/AU
L9 0 L7 AND WU, Z?/AU

=> s l7 and zhao, z?/au
7927 ZHAO, Z?/AU
L10 0 L7 AND ZHAO, Z?/AU

=> s l7 and hartnett, j?/au
222 HARTNETT, J?/AU
L11 0 L7 AND HARTNETT, J?/AU

=> d l7, ibib abs hitstr, 1-2

L7 ANSWER 1 OF 2 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2005:1288063 HCAPLUS
DOCUMENT NUMBER: 144:36364
TITLE: Bicyclic compounds
INVENTOR(S): Hirai, Miki; Kusama, Mari; Hosaka, Toshihiro; Kohnomi, Shuntarou
PATENT ASSIGNEE(S): Tanabe Seiyaku Co., Ltd., Japan
SOURCE: PCT Int. Appl., 68 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2005115984	A2	20051208	WO 2005-JP10287	20050530
WO 2005115984	A3	20060302		

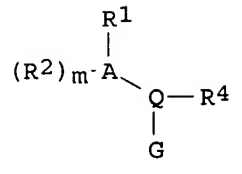
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.:

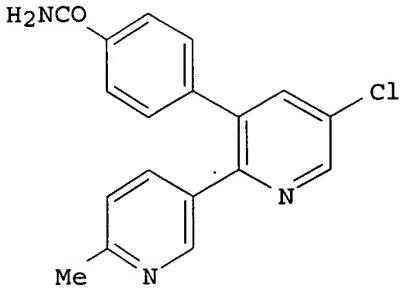
JP 2004-160660	A	20040531
JP 2004-191849	A	20040629
US 2004-584142P	P	20040701
JP 2004-348136	A	20041201

OTHER SOURCE(S): MARPAT 144:36364
GI

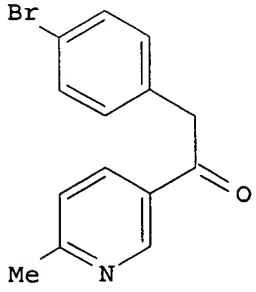
Updated Search



I



II



III

AB Heterocyclic compds. I [Q = pyridine or pyrimidine; A = benzene or heteroarom. ring; G = ring B optionally substituted with R3, or amino optionally substituted by one or two selected from the group consisting of alkyl, aralkyl and cycloalkyl; ring B = benzene, heterocyclic ring, cycloalkane or cycloalkene; R1 = CON(R6)R5, CON(R6)OR5, CONHN(R6)R5, COON(R6)COR5, CON(R6)SO2R5, COR5, CO2R5, CN; R2 and R3 may be the same or different from each other, and each = CN, NO2, OH, alkoxy, halo, carboxyl, etc.; m = 0, 1 or 2; R4 = H, CN, OH, halo, alkoxy, carbamoyl, etc.; R5 and R6 may be the same or different from each other, and each = H, an optionally substituted alkyl, cycloalkyl, aryl, heterocyclic, alkoxycarbonyl, or R5 and R6 may form an optionally substituted heterocyclic ring in combination with atoms to which they are bonded] and pharmaceutically acceptable salt were prepared as calcium-activated K channel opener useful for treatment of pollakiuria, urinary incontinence, chronic obstructive lung disease and prophylaxis. Thus, compound II was prepared via heterocyclization reaction of III with Vilsmeier agent, and showed a relaxation effect on K-induced contraction of isolated urinary bladder.

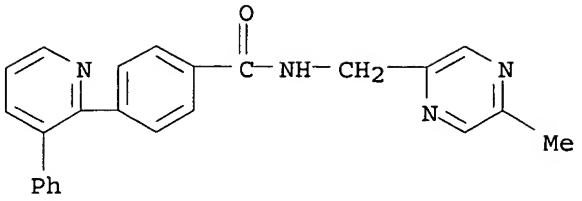
IT 870723-07-2P 870723-08-3P 870723-09-4P
RL: PAC (Pharmacological activity); SPN (Synthetic preparation); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(preparation of heterocyclic compds. as calcium-activated K channel opener for treatment of pollakiuria, urinary incontinence, chronic obstructive lung disease and prophylaxis)

RN 870723-07-2 HCAPLUS

CN Benzamide, N-[(5-methylpyrazinyl)methyl]-4-(3-phenyl-2-pyridinyl)-, monohydrochloride (9CI) (CA INDEX NAME)

Updated Search

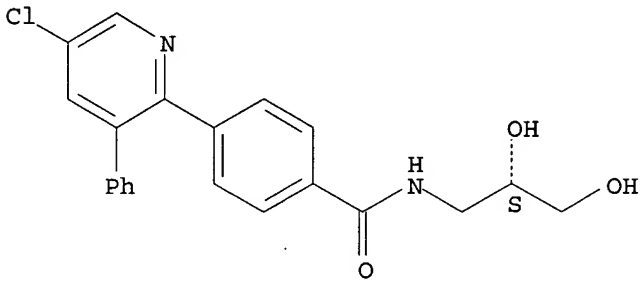
10554187



● HCl

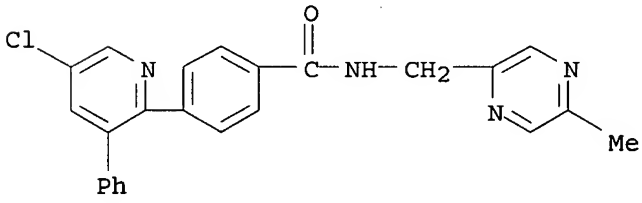
RN 870723-08-3 HCAPLUS
CN Benzamide, 4-(5-chloro-3-phenyl-2-pyridinyl)-N-[(2S)-2,3-dihydroxypropyl]-, monohydrochloride (9CI) (CA INDEX NAME)

Absolute stereochemistry.



● HCl

RN 870723-09-4 HCAPLUS
CN Benzamide, 4-(5-chloro-3-phenyl-2-pyridinyl)-N-[(5-methylpyrazinyl)methyl]-, monohydrochloride (9CI) (CA INDEX NAME)



● HCl

L7 ANSWER 2 OF 2 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2003:737516 HCAPLUS
DOCUMENT NUMBER: 139:257284
TITLE: Cathepsin cysteine protease inhibitors and their therapeutic use

Updated Search

10554187

INVENTOR(S): Bayly, Christopher I.; Black, Cameron; Leger, Serge; Li, Chun Sing; McKay, Dan; Mellon, Christophe; Gauthier, Jacques Yves; Lau, Cheuk; Therien, Michel; Truong, Vouy-Linh; Green, Michael J.; Hirschbein, Bernard L.; Janc, James W.; Palmer, James T.; Baskaran, Chitra

PATENT ASSIGNEE(S): Merck Frosst Canada & Co., Can.; Axys Pharmaceuticals, Inc.

SOURCE: PCT Int. Appl., 282 pp.
CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.		DATE
WO 2003075836	A2	20030918	WO 2003-US6147		20030228
WO 2003075836	A3	20040715			
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW					
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG					
CA 2477657	AA	20030918	CA 2003-2477657		20030228
AU 2003219953	A1	20030922	AU 2003-219953		20030228
US 2003232863	A1	20031218	US 2003-377377		20030228
EP 1482924	A2	20041208	EP 2003-716238		20030228
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK					
BR 2003008208	A	20050111	BR 2003-8208		20030228
CN 1638757	A	20050713	CN 2003-805181		20030228
JP 2005526753	T2	20050908	JP 2003-574112		20030228
US 2005240023	A1	20051027	US 2004-505796		20040825
NO 2004004207	A	20041124	NO 2004-4207		20041004
PRIORITY APPLN. INFO.:			US 2002-361818P	P	20020305
			US 2002-408704P	P	20020906
			WO 2003-US6147	W	20030228

OTHER SOURCE(S): MARPAT 139:257284

AB This invention relates to cysteine protease inhibitors R7(D)nCR6R7NR8CR3R4C(:O)NHCR1R2CN (R1-4 = H, (substituted)C1-6-alkyl or C2-6-alkenyl; R1 and R2 or R3 and R4 may be take together with the C atom to which they are attached to form a (substituted)C3-8-cycloalkyl or heterocyclic ring; R5 = H, (substituted)C1-6-alkyl; R6 = (substituted)aryl, heteroaryl, C1-6-haloalkyl, arylalky, heteroarylalkyl; D = (substituted)C1-3-alkyl, C2-3-alkenyl, C2-3-alkynyl, aryl, heteroaryl, C3-8-cycloalkyl, heterocyclyl; R7 = H, (substituted)C1-6-alkyl, C2-6-alkenyl, C2-6-alkynyl, C1-6-alkyloxy, etc.; R8 = H, C2-6-alkyl) including but not limited to, inhibitors of cathepsins K, L, S and B. These compds. are useful for treating diseases in which inhibition of bone resorption is indicated, such as osteoporosis.

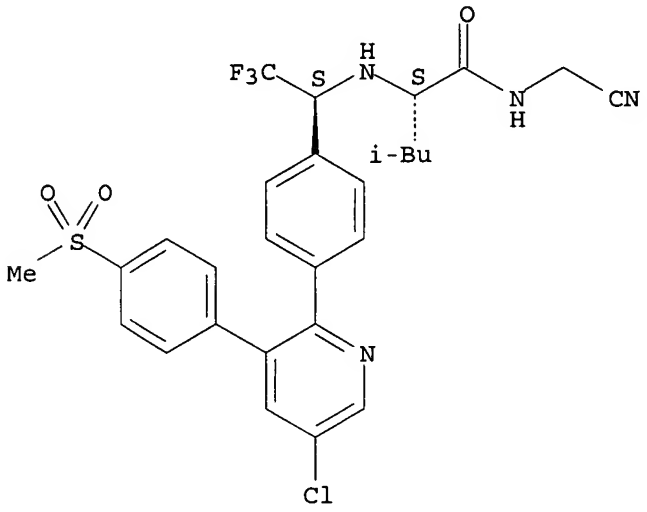
IT 603140-97-2P
RL: SPN (Synthetic preparation); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)

Updated Search

10554187

(cathepsin cysteine protease inhibitors and their therapeutic use)
RN 603140-97-2 HCAPLUS
CN Pentanamide, 2-[[(1S)-1-[4-[5-chloro-3-[4-(methylsulfonyl)phenyl]-2-pyridinyl]phenyl]-2,2,2-trifluoroethyl]amino]-N-(cyanomethyl)-4-methyl-, (2S)- (9CI) (CA INDEX NAME)

Absolute stereochemistry.



=> file caold

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	25.50	194.60

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	-3.00	-3.00

FILE 'CAOLD' ENTERED AT 14:42:25 ON 02 OCT 2006
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.
PLEASE SEE "HELP USAGETERMS" FOR DETAILS.

FILE COVERS 1907-1966
FILE LAST UPDATED: 01 May 1997 (19970501/UP)

This file contains CAS Registry Numbers for easy and accurate substance identification. Title keywords, authors, patent assignees, and patent information, e.g., patent numbers, are now searchable from 1907-1966. TIFF images of CA abstracts printed between 1907-1966 are available in the PAGE display formats.

New CAS Information Use Policies, enter HELP USAGETERMS for details.

This file supports REG1stRY for direct browsing and searching of all substance data from the REGISTRY file. Enter HELP FIRST for more information.

Updated Search

10554187

```
=> d his

     (FILE 'HOME' ENTERED AT 14:36:42 ON 02 OCT 2006)

     FILE 'REGISTRY' ENTERED AT 14:39:04 ON 02 OCT 2006
L1                STRUCTURE UPLOADED
L2              1 S L1
L3             36 S L1 FULL

     FILE 'HCAPLUS' ENTERED AT 14:41:10 ON 02 OCT 2006
L4              4 S L3
L5              4 S L3/THU
L6              2 S L5 AND DUGGAN, M?/AU
L7              2 S L5 NOT L6
L8              0 S L7 AND LINDSLEY, C?/AU
L9              0 S L7 AND WU, Z?/AU
L10             0 S L7 AND ZHAO, Z?/AU
L11             0 S L7 AND HARTNETT, J?/AU

     FILE 'CAOLD' ENTERED AT 14:42:25 ON 02 OCT 2006

=> s l3
L12             0 L3
```

Updated Search